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An Address.[1]

BE IT ENACTED.

By A. McL. MILLAR,

Retiring President of the Tasmanian Branch of the British Medical Association.

THE year 1958 marked the centenary of the General Medical Council of Great Britain. Events leading up to the establishment of this Council, by the *Medical Act* of 18'8, and its subsequent history were reviewed in an article by F. N. L. Poynter, B.A., Ph.D., in the *British Medical Journal* of November 22, 1958.

It is my intention today to recall a few details from Dr. Poynter's story of this body, which from a stormy inception now exerts a profound interest in and control over 90,0000 practitioners in the United Kingdom, and to pass on to a very brief review of the multiple and varied State medical Acts, which are the instruments of practice of a comapartively small number of practitioners in Australia, and to comment particularly on the functions and powers of the various State Medical Boards (or, as it is called in Tasmania, the Medical Council).

[1] Delivered at the annual meeting of the Tasmanian Branch of the British Medical Association on March 7, 1959.

The passing of the *Medical Act* of 1858 was the first practical step towards the development of the united and well-organized profession of a uniformly high standard which exists today. The declared purpose of the Act was to enable the public to distinguish qualified from unqualified practitioners, quackery and charlatanism being then rife. It was appreciated early that the Act would not achieve its object unless "the legally qualified practitioner shall be a well qualified practitioner".

From its inception there were two primary objects: firstly, to insist on an adequate educational qualification; and secondly, to police and punish malpractice. Thus it is of importance to appreciate that the legally defined purpose of the Act was to safeguard the public interest, and not to advance the welfare of the medical profession. These aims have remained unchanged throughout the century, and are also embodied, sometimes vaguely and sometimes in great detail, in the various Australian State medical Acts.

It is, then, surprising to realize that the vast majority of the public, in Australia at any rate, remain quite unaware of the functions and powers or even of the existence of the Medical Boards, whose very being is for their benefit and protection, and have come to regard the British Medical Association as the wielder of the baton in the case of misdemeanour of a practitioner. As the Association rarely finds it necessary to mete out the punishment which the complainant considers just and adequate, there is often an unnecessary

deleterious effect on our public relations. Further in regard to this point, it is relevant to make some comparison with the wording of the ethical rules of the Australian State Branches of the British Medical Association, which indicate that an ethical breach is regarded as conduct which is detrimental to the honour and interests of the medical profession or the Association; although it is implied in most instances, there is no specific reference to the rights or welfare of the public.

During the past year, I consider on reflection that the most important parochial business which your Branch Council had to consider was the request by a majority of the members that the Medical Council of Tasmania should seek an increase in its powers by an amendment of the Act. I am ashamed to admit that, until our delegation waited on the Council (Board), I had never read the *Medical Act* of Tasmania, but am now convinced that it is the first responsibility of every practitioner, on being registered, to study the medical Act of the State in which he practises. After this, and for the purpose of this address I have made a superficial and lay reading of the Medical Acts of all the other States, and find it a revelation that such varying Acts are necessary to control the practice of homologous groups of practitioners and to protect the rights of homologous groups of citizens who happen to reside in different geographical areas.

In Tasmania we practise fairly harmoniously, and I think efficiently, under the control of a medical Act which is virtuous in its very brevity, and we flourish despite the absence of clauses, as in the New South Wales Act, which define the dimensions of a brass plate and the size of its lettering, and in meticulous detail the type, size and illumination of the outside lights we may use. It is granted that in such aspects the Association's ethical rules have a curbing influence; but surely we should make up our minds on a Commonwealth basis as to who is the responsible power.

Let me present you with a series of disjointed facts, derived from the Acts, which will serve to illustrate the unusual situation present in Australia. The actual constitutions of the State Boards vary considerably in their size, qualifications for membership and terms of office. For example, Queensland's seven members comprise the Director-General of Health, who is president, three members nominated by the Minister to represent the Government, and three members nominated by the Association. South Australia stipulates five members, three being nominated by the Minister, one by registered practitioners and one by the Council of the University of Adelaide. The New South Wales Board consists of not less than seven or of more than nine members, one of whom is appointed by the Senate of the University of Sydney, and one who is a practitioner of five years' standing appointed by the British Medical Association. Victoria and Tasmania are somewhat similar, inasmuch as each simply lays down the requisite number without specifying qualifications, and whereas other States seem keen to exclude the mentally deficient, the bankrupts and those with criminal records, these States make no such provision in their Acts.

So much for the constitution of the various Boards. Their first duty, as regulated by the Acts, is in relation to the registration of practitioners, and here there is a basic uniformity apparent despite a gross disparity of verbiage. The underlying principle is that all registered practitioners shall measure up to the academic standard of an Australian university qualification, and in spite of any *Alma Mater* allegiances we may have, there is little doubt that these are uniform and of a high standard. In addition to academic qualifications is the necessity of twelve months' practice in an approved hospital or institution, and in regard to this we once more find a lack of uniformity. For example, the New South Wales Act has a clause to waive this if there is physical incapacity to comply with it, and will accept some other training in lieu thereof. Surely this is anomalous, in that it does not appear in other State Acts, just as the Victorian Act gives credit to service in the Royal Australian Army Medical Corps for two years between 1940 and 1946, whereas other States do not. The various State Acts simply bristle with a multiplicity of contingencies in relation to registration, and it is obvious that, if they are necessary in one State, they are necessary in all and vice versa, with but few minor exceptions. Before passing on from the subject of registration, I would remark that New South Wales appears to be the only State in which reciprocity is demanded. That is, if a University of Sydney graduate is not accepted in some other part of the Empire, then graduates of that part of the Empire will not be registered to practise in New South Wales.

The laws enacted in the different States, in relation to the registration of persons with foreign degrees or diplomas, are fairly uniform, with the exception that Tasmania distinguishes itself in that it legislates for the conferring of a special licence to practise. These licences are restricted in number, are entailed with restrictions as to the nature and site of practice, and allow the licentiate to designate himself Tasmanian licentiate in medicine or in surgery, or in medicine and surgery as the case may be.

It is perhaps understandable that in Australia, because of its geography and its distribution of population, it has been necessary to bring in slight variations or modifications of qualifications for registration, in order to provide an adequate medical service to the public, and so, for instance, we have some States making provision for restricted regional registration. However, it is quite impossible on any State, geographical, social or economic grounds to rationalize the lack of uniformity in regard to the disciplinary powers of the various Boards and their methods of implementing these powers.

To revert for a moment to Dr. Poynter's article, it is historically important to appreciate that the British Medical Council was the first statutory tribunal of its kind with quasi-judicial powers over a whole profession, and also, in its early years, there was only one punishment it could inflict; that was erasure from the Register, and further, there was no right of appeal against such a decision.

Let me say that it is obvious that political or legal control is rarely necessary for the conduct of efficient and ethical practice in Australia; but, as there certainly must be some supervision, then let it be uniform throughout, instead of the existing higgledy-piggledy arrangement. Perhaps in other States the desirability of this may not be so evident, as the majority of practitioners in Australia qualify in their home States, and register and practise there. We in Tasmania have all qualified elsewhere. All of us have been registered in at least one other State or country, and have thus—though many will be unaware of it—owed obedience to quite different regulations, and have been responsible to different medical boards with vastly differing powers.

It appears from the Act that the South Australian Medical Board in itself has no disciplinary powers whatsoever, and that offences against the Act are to be dealt with by civil court action. Western Australia goes to the other extreme, as a reading of the Act indicates that the Board in itself has, after inquiry, the power of erasure or suspension for such time as it deems fit, reprimand or the imposition of a fine. There is, however, the right of appeal to a Judge of the Supreme Court.

The Victorian Board may in its own right erase a name from the Register, but I could find nothing in the Act which allows for an appeal to the Supreme Court, and furthermore, there is scant reference to other powers of the Board. This, in an otherwise voluminous Act, is either surprising in its omission or complimentary to the standards of practice in that State.

The disciplinary powers of the Council (Board) in Tasmania are liberal, in that it has the power to deal with complaints of improper conduct in a professional respect, and itself will charge a person and shall hear and determine the charge. If the Council is satisfied that a charge is sustained, it may reprimand the practitioner or fine him up to £200, or if it is satisfied that the practitioner has been guilty of improper conduct

sufficiently grave to constitute infamous conduct, it may make application to the Supreme Court. Further, on the motion of the Council, a person may be removed from the Register by order of the Supreme Court, or the Court may order suspension of his registration for such time as it thinks proper.

The Queensland Act throughout is a model document, and this is exemplified particularly in relation to the sections dealing with misconduct and the method of dealing with it. The practitioner in Queensland, having studied his State Medical Act, can have no doubts as to his obligations and responsibilities to the law and to the public, as these are laid down in detailed clarity. Pursuing a middle course, Queensland has, in addition to its Medical Board, a Medical Assessment Tribunal "for the better control of medical practitioners and for the better determination of prescribed matters having a medical element". This Tribunal is constituted by a Judge of the Supreme Court, to which the Governor in Council appoints two medical practitioners who sit as assessors, one nominated by the Government and one nominated by the British Medical Association, and it is the duty of the assessors to advise the Judge in the proper determination of any fact which may arise. The Board refers to the Tribunal any question as to the conduct or qualifications of any medical practitioner which, in the opinion of the Board, requires investigation in the public interest. However, if after investigation the Board is of the opinion that the evidence has not sufficiently established a prima-facie case, it dismisses the case, or where it considers the import of the matter is not sufficiently serious to warrant a charge before the Tribunal, it may fine or reprimand the practitioner. The Tribunal has the power to erase a practitioner from the Register, suspend him for such time as it shall specify or fine him up to £100. The possibility of unnecessary and undesirable publicity in these matters is abhorrent to all, and this is anticipated in the Queensland Act, which directs that no order, determination or finding shall be made public except at the discretion of the Board. This is an extremely important proviso.

Although I am quite incompetent to say that the Queensland Act is an ideal, I dare to say that it is a vast improvement on other State Acts, and that it might well be used as a basis for a uniform set of laws throughout the Commonwealth. Compared with the powers outlined above, the Association has very few indeed, and what poor powers it has are extremely difficult to implement.

I must now attempt to justify my action in presenting to you these translations. If you care to read again the Memorandum of Association of the British Medical Association, you will find that, prosaically enough, the first two paragraphs state that the name of the Association is the British Medical Association, and that the registered office of the Association is to be in England. The third paragraph, which occupies the bulk of the Memorandum, then goes on to detail eight objects of the Association.

The first three objects are related to the advancement of the medical and allied sciences, the holding of meetings and the publication of a journal and papers. The remaining objects are concerned with finance, property and similar inconsequential matters. The practitioner or the patient, depending on the point of view, is thus under the aegis of two distinct bodies. One body is avowedly primarily scientific and the other avowedly disciplinary. The patient is acutely aware of the existence of the former body, often to that body's detriment in the matter of public relations, yet is, in the main, unaware of the existence of the latter body.

It is my contention, today, that practitioners should endeavour to inculcate into the public mind, by judicious advice when the occasion arises, the true objects of the Association, and to divert appropriate complaints to the proper channel, and so help to maintain public esteem of the British Medical Association on the high level which is its right.

PERIPHERAL VENOUS OBSTRUCTIONS.[1]

By JOHN LOEWENTHAL,
Professor of Surgery, University of Sydney, Sydney.

IT is surely fair to say that we take our veins for granted. After all, who is interested in a drainage system of any sort until it goes wrong? All the glamour and publicity is lavished on the supply side; Sydneysiders know what a howl of rage arises when water restrictions are imposed in spring and summer; but a blocked sewer outfall passes unnoticed.

The comparative incidence of lesions on either side of the vascular tree is greater on the venous side—at least so far as the limbs are concerned. In a busy peripheral vascular clinic attended by an average of 60 to 70 patients per week, 20% only have purely arterial disease. That venous conditions are, on the whole, less lethal than arterial ones is freely conceded, but the morbidity and chronic invalidism resulting from them are certainly no less. Their greater incidence means that they are a source of much social and economic loss; but this damage can be minimized by active and vigorous management of the acute thrombotic phase.

The Acute Thrombotic Phase.

In a sensitively adjusted mechanism like the vascular tree, affection of any portion of it invariably leads to widespread effects. A mechanism which can accurately control the oxygen demands of tissues—which may be either at rest or playing a Beethoven symphony or even running a mile in four minutes—must have efficient control on both the input and the output side, and this control must necessarily be highly integrated and coordinated. Therefore, the presence of a thrombus in a vein produces effects of more significance than would arise from simple blockage of outflow from the part. A simple block may be produced by ligation of the femoral vein, a procedure once used in normal legs as prophylaxis against post-operative thrombo-embolism. This causes minimal œdema of the limb, sometimes of such minor degree as to be scarcely detectable. A thrombus in the same area, affecting iliac and femoral vein, is a much more damaging and serious affair; the pain and swelling of the limb and general systemic disturbance are often extreme, with the development classically of a "white leg". This phlegmasia alba dolens probably results from uncontrolled reflex arteriolar and possibly venous spasm, mediated through the vascular autonomics; it is true that in some earlier cases, if the reflex arc is interrupted by paralysis of the lumbar sympathetic chain, rapid and dramatic reversal of the condition occurs.

This type of reaction is seen in extreme degree in the condition of phlegmasia cærulea dolens which may proceed to "venous gangrene".

A female patient, aged 35 years, presented with a swollen, painful right leg, and signs of superficial thrombophlebitis. She had a mass in the pelvis which was subsequently shown to be a malignant ovarian cyst when removed at operation. The phlebitis subsided with anticoagulant therapy, but soon recurred extensively in the deep veins of the same limb, with onset of gangrene in the toes and the sole of the foot (Figure I). At this stage all the peripheral arterial pulses were full and bounding, and the condition began to appear in the great toe of the other foot. Full and continued heparinization turned the thrombotic tide, and after some weeks the gangrene had become demarcated (Figure II). After a further period this area was excised.

One can only guess at the nature of the mechanism which produces peripheral gangrene in sheets or layers in the presence of a full arterial inflow. Is it extreme peripheral stasis or some proximal and unsuspected shunt, or widespread arteriolar constriction? Be that as it may,

[1] Read at a meeting of the Section of Surgery, Australasian Medical Congress (B.M.A.), Tenth Session, Hobart, March 1 to 7, 1958.

this type of case indicates the profound effect venous occlusion may have on other tissues, and it would seem logical to assume that the same process occurs in lesser degree in other cases of venous thrombosis.

Effects of Venous Thrombosis.

Immediate Effects.

The quiet development of a thrombus in a vein leads to interesting local changes. The partial or complete occlusion of the trunk by thrombus causes reduction of the lumen of the vein itself, and of its tributaries. It is likely that an element of venospasm also occurs and further reduces the total cross-sectional area available for outflow. This reduces the mean lineal velocity of flow through the part, causing comparative stagnation. The effect of this, and of the developing phlebitis in the wall of the vein, is to produce margination of leucocytes and platelets along the vein wall. The platelets by aggregation become more fragile and sticky, so that there is a greatly increased local concentration of thromboplastin liberated from large numbers of these unduly fragile thrombocytes. The slowly-flowing blood in the narrow venous bed then passes into a "sludge state"—an intermediate phase in the change from the hydrosol fibrinogen to the hydrogel fibrin. This state is marked by a slow rate of flow and a high local concentration of thromboplastin. From this point of more or less equilibrium there may be a sudden mass conversion to fibrin with rapidly spreading thrombosis, or, under different circumstances, a reversal of the reaction and the reconversion of the sludged blood to soluble fibrinogen.

Throughout the body, the state of any enzymatic action at a given moment is the sum of the effects of the opposing factors operating. In the complicated enzymatic chain which constitutes coagulation the main opposing agent as yet known is heparin. As might be expected on philosophical grounds, it attacks this chain-reaction at a variety of points—it is an anti-prothrombin and an anti-thromboplastin, and is known to reduce both the stickiness and fragility of platelets. There is some evidence that it can reduce acute vascular spasm. Therefore, its primary importance in the management of thrombotic states cannot be over-emphasized. By its effect on platelets, local thromboplastin and thrombin concentrations are reduced, and this, together with relief of venospasm and the clearing of sludged blood, reduces local stagnation, improves outflow, reduces œdema significantly and rapidly lessens local pain. In essence, it effectively changes the state of enzymatic equilibrum to one of resolution.

The clinical significance of this is clear: early full, active heparinization of the patient with venous thrombosis is imperative if mortality, morbidity and local sequelæ are to be reduced.

We use heparin in accordance with the following standards.

FIGURE I.
Phlegmasia cærulea dolens, developing phase.

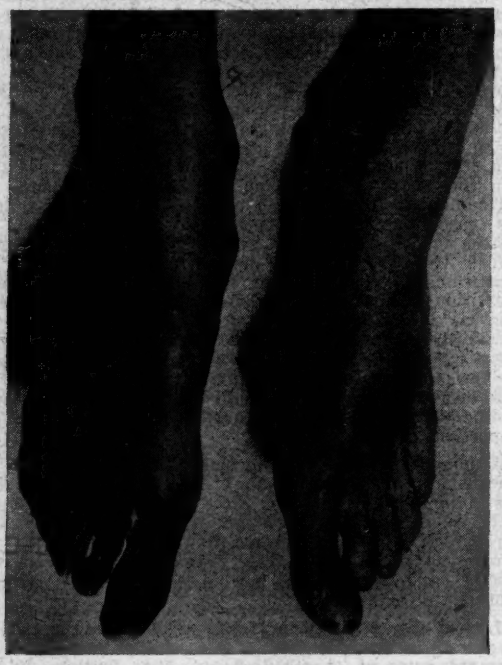

FIGURE II.
Phlegmasia cærulea dolens. Venous gangrene demarcated. Peripheral pulses full and bounding

1. Intravenous administration is the only effective method. Subcutaneous and intramuscular injections can lead to huge, extremely distressing hæmatomata, and because of variations in absorption rate, are largely capricious and unreliable in effect.

2. Capillary clotting times are too inaccurate to justify their use as a means of determining heparin dosage.

3. The average adult can be fully heparinized by 10,000 units injected intravenously every eight hours. In some cases heparin can be given intravenously in a dosage of 5000 units every four hours, or even continuously. A daily intravenous dose of 30,000 units produces a full heparin effect, and in our hands over many years now is safe without any laboratory control.

4. The efficiency of other anticoagulants given as a short-term or long-term measure is far from proven, and in my opinion, they should be given only after the physiological anticoagulant has been exhibited for long enough to have a full effect. We continue to give heparin until all local tenderness over the affected vein has disappeared —a period which may extend to two or three weeks.

5. When in doubt, give heparin. Such a doubt most commonly occurs when a patient complains of a sudden pain in the chest a few days after operation. Neither the anæsthetist nor the radiologist nor the physician can assure us that the damage is in an area of atelectasis rather than a pulmonary infarction. Quite often, if 10,000 units of heparin are injected intravenously, there is a significant reduction in the degree of pain within 10 to 15 minutes. This can be taken as good circumstantial evidence of a thrombo-embolic origin to the condition, and anticoagulant therapy should be continued. Frequently, overt tenderness and swelling of a calf will be noted a day or two later to clinch the matter.

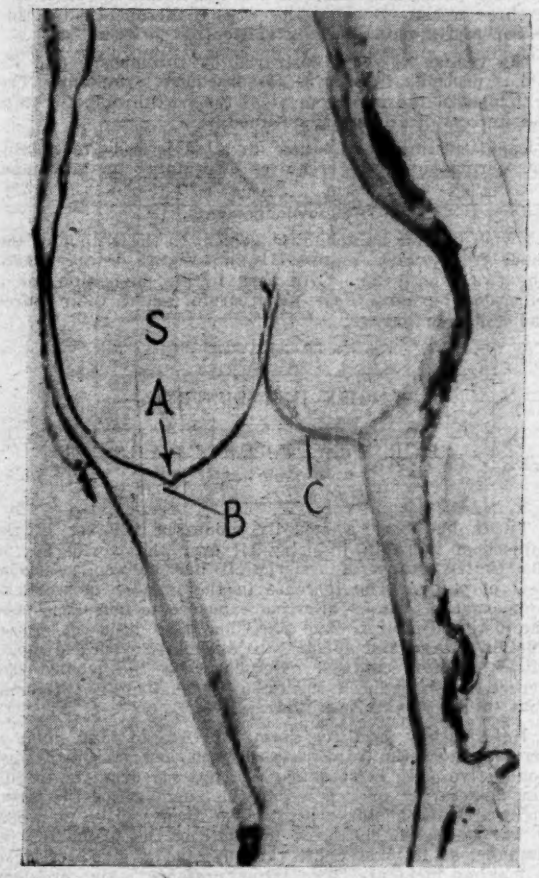

FIGURE III.
Normal venous valve (after Edwards).

Remote Effects.

The structure which ultimately suffers most from an intravenous thrombus is the venous valve. The great majority of clotted veins become recanalized, but their valves invariably suffer permanent damage. The normal valve is a simple bicuspid arrangement (Figure III), which can be seen clearly in a phlebogram (Figure IV). As a result of organization of a thrombus, the valve cusps become either digested away or stenosed, or are caused to adhere to the recanalized vein wall (Figure V).

The post-thrombotic syndrome in the lower limbs—pain, swelling, ulceration and eczema—varies greatly in degree in different patients, depending on circumstances, such as the age and build of the patient and the extent and management of the thrombus. In one person pain is predominant, in another swelling, or there may be mixtures of these in every possible variation. It seems likely that it is the fate of the involved valves which is the critical factor, but this is excessively hard to demonstrate. Man is the only animal who stands solely on his back legs, and used as an experimental animal to prove this point, he is unsatisfactory.

Treatment.

Some of the guiding principles used in our clinic in the management of this common condition are as follows.

1. The acute phase is treated fully and energetically. We heed the words: "The physician who attends a patient with a thrombosis in the leg is presiding at the commencement of a lifelong tragedy."

2. "Compensatory" varices secondary to a deep vein thrombosis rarely occur. The idea that operative cure

FIGURE IV.
Phlebogram, showing normal venous valve in thigh segment.

must never be practised for varicosities in a limb which has once suffered a deep clot is quite erroneous. The varices should be examined by the ancient method of William Harvey to see whether they fill from above or below. Almost invariably it will be from above, in which case they are doing no more than embarrassing the circulation, and can be safely ligated.

3. Post-thrombotic pain is sometimes found accompanied by only slight swelling, and can usually be relieved by sympathetic release. The pain is characteristic—it is full and "bursting" in nature; when very severe, it may come on after walking a fairly standard distance and so simulates ischæmic claudication. However, it is easily distinguished, as the peripheral pulses are present and normal and the pain is quickly relieved by elevation, whereas ischæmic pain responds best to dependency. This constitutes a small group of patients who can be salvaged by sympathectomy. Relief of the pain leads to improved muscle action and some small reduction in swelling.

4. The chronically swollen limb has to be treated with patience and persistence. Every effort must be made to

keep the swelling controlled. Weight reduction when necessary is fundamental. Properly fitted elastic supports, avoidance of periods of prolonged standing, sleeping with the foot of the bed raised, and regular periods of rest with elevation of the limb during the day all play their part. These limbs are prone to recurrent attacks of cellulitis, and the patient is often able to recognize from experience the characteristic prodomata of such an attack. The infection usually responds well to chemotherapy, especially if it is used early in the train of events. We therefore supply the patient with a small store of appropriate antibiotics so that delay in beginning treatment is avoided. It is clear that each attack further damages the venous and lymphatic outflow, so all efforts are made to abort it and so to minimize the damage.

FIGURE V.
Venous valve cusps involved in thrombus (after Edwards).

5. It seems axiomatic that once a lower limb has suffered a deep vein thrombosis, it is always susceptible to ulceration of a peculiarly painful and intractable type. Even though the residuum of the thrombus may be minimal swelling, the limb is still in jeopardy. Therefore the slightest break in continuity of the skin, especially in the distal half of the leg, should be treated seriously, with, if necessary, a period of bed rest.

Once ulceration has become established, a vast literature bears eloquent evidence, not to the difficulty in healing the ulcer, but to that encountered in keeping it healed. We seek out and destroy all factors leading to peripheral stasis whether they are superficial varices or deep perforators; we sterilize granulations carefully, excise the underlying scar, and inlay skin grafts accurately; in some few cases we offer sympathectomy to help stabilize the graft, and to dry the surrounding sodden and eczematous skin; all the arts of physiotherapy, reablement, rehabilitation, physical and even mental support are used —and we still have recurrences. The lesson is clear: the underlying vascular abnormality remains, and the keynote of success is "maintenance". "Once an ulcer—always an ulcer" applies as justifiably to the post-phlebitic limb as to the scarred duodenum. At least in the leg, recurrence can be seen as soon as it commences, and much suffering and loss can be avoided by its early effective treatment. In many ways this is a social as much as a medical problem, but "the price of liberty is eternal vigilance".

Summary.

Venous lesions are commoner than arterial lesions and a source of great social and economic loss.

A venous thrombosis causes serious effects beyond simple obstruction to outflow. Actual gangrene can occur from purely venous obstruction, and the mechanism causing this extreme effect probably exists to lesser degree in all cases.

The immediate local results of an intravenous clot is slowing of the rate of flow, increased thromboplastin and "sludged" blood. Mass clotting may ensue, or resolution can occur. Heparin, the physiological anticoagulant, opposes this process at all points; it should be given intravenously until all local tenderness has subsided—up to two or three weeks. It can be used as a therapeutic test for post-operative pulmonary infarction.

The venous valve is destroyed by thrombosis, and its fate is probably the source of subsequent symptoms. The principles of the management of the post-thrombotic state and its complications are outlined.

Rapid healing of the ulcerated leg is easy to obtain, but permanent relief requires continuing maintenance.

Acknowledgements.

I wish to offer an acknowledgement to the author of the paper from which Figure III and Figure V are taken. The two illustrations have been in my possession for a long time, but despite the most careful search their source has not been located.

NOISE IN INDUSTRY.

By R. WILLIS, F.R.C.S., F.R.A.C.S.,
Melbourne.

THERE has been a steadily mounting interest in the subject of noise in industry all over the world, but in the United States of America it has become a special field of study. The increase in the number of workers' compensation claims following exposure to noise has been the main stimulus; but there has also been the introduction of new and noisier jobs associated with the repair of jet aircraft engines. Emphasis on the medico-legal aspect has introduced a number of challenging problems that industry must face and resolve.

Hearing loss resulting from exposure to loud noise is termed "acoustic trauma", and such loss may be either conductive (i.e., blast damage to the ear drum or middle ear structures) or perceptive (i.e., damage to the nerve structures in the inner ear). The former gives no worry either in the diagnosis or in allotting the blame for the damage, as it is of acute type, related to one particular damaging incident. The latter presents many problems and hence is the one discussed here.

Perceptive or inner ear deafness may result from prolonged exposure to loud noise such as is found in industry, or from brief exposure to intense noise such as gunfire. Minimal exposure produces a temporary deafness known as "auditory fatigue", and we have all no doubt experienced this at some time. Either after a noisy air trip or after being too near a friend's shotgun muzzle, we find ourselves with "fuzzy" hearing and with a ringing in our ears. The duration of this fatigue depends on the severity of the noise. If we are exposed beyond a certain limit it ceases to be a temporary problem and permanent damage shows up.

Some persons show undue auditory fatigue on exposure to not very intense noise, and we class them as susceptibles. Ideally we should exclude all such people from noisy industry, as they will certainly develop permanent deafness if they continue working in noise. We have tests which will fairly accurately pick out these people before they reach the stage of permanent damage.

The pathological change in perceptive deafness begins in the outer hair cells of the organ of Corti in the basal turn of the cochlea. This produces the typical hearing loss for the high-frequency tone of 4000 cycles per second. On the audiogram this shows as the "4000 dip" and is almost proof positive that noise (or blast wave) has been the cause.

The frequencies we are concerned with in human speech are 500 to 3000 c.p.s. Therefore, provided that the hearing loss due to noise does not involve anything more than the "4000 dip", the person concerned does not know that he is deaf. However, if the exposure to noise continues, frequencies on either side of 4000 c.p.s. become progressively involved, and soon the upper limit of the speech range is encroached upon. It so happens that we tend to hear most of the consonants at the upper end of the speech scale, and it is upon the consonants, particularly

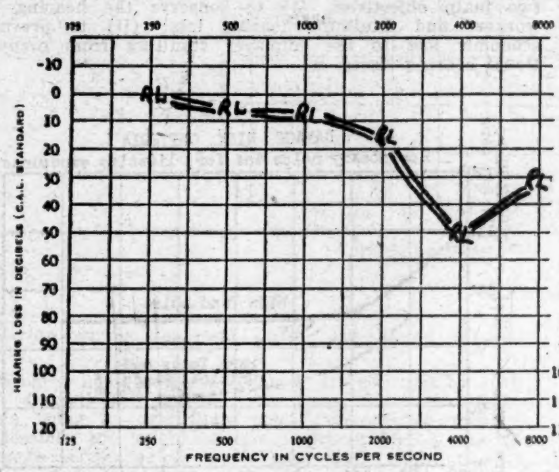

FIGURE I.

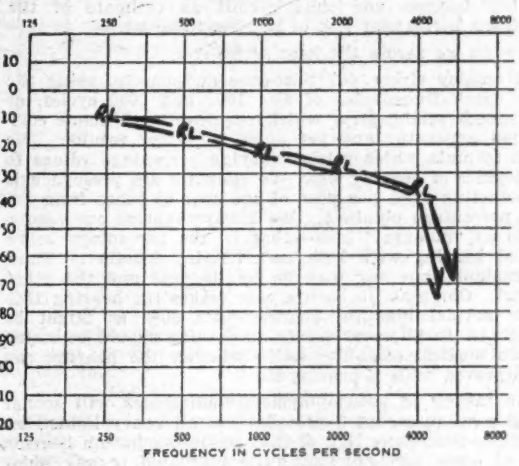

FIGURE II.

at the beginning and end of a word, that we rely for intelligibility of speech. Therefore, the first sign that acoustic trauma is affecting the hearing of speech is that the person concerned can hear people talking but cannot distinguish what exactly is being said. Figure I will illustrate a fairly typical audiogram from a case of acoustic trauma that is causing worry because it has encroached upon the speech area.

As everyone ages, a form of deafness occurs which we know as "presbyacusis". This is comparable with presbyopia, the gradual failing of near vision with advancing age. Figure II shows the audiogram from such a case, and you will note that there are many features in common with the noise-damage case. For this reason, as we shall see later, due allowance must be made for age in all assessments of hearing loss due to industrial noise.

In 1953 an interesting report of the deafness encountered in intense noise fields was prepared by Dr. Luigi Bernabei, of Milan. He studied drillers of borax wells in Tuscany. Such a well begins as a "gusher" when the gas is struck below ground. Until the gusher is controlled, the drillers must work for varying periods in noise above 130 decibels in intensity. Some protection of the men is obtained by filling their ears with wood putty. Bernabei noted some of the features of exposure to such an intense noise; deafness was immediate and permanent, and affected a wide range and not merely the 4000 c.p.s. point; tinnitus, or ringing in the ears, was a permanent worry to over a third of the workers; vertigo, or unsteadiness due to inner ear disturbance, was often pronounced, and occasionally a worker had to be withdrawn from the job completely because of it; and abdominal discomfort, indisposition, thoracic constriction and muscular tremor were attributed to the intense vibrations that accompanied the noise. He found, interestingly enough, that the noise did not affect the worker's productivity on the job.

This last observation is in keeping with that of Kryter, who in 1950 denied most reports that noise adversely affects work output. Steady and expected noise has no deleterious effect and may even improve the performance of work by insulating the worker from distractions. We do know, however, that certain high-frequency noises have an intense annoyance effect.

The medico-legal aspects of industrial deafness are exercising many legal minds at the moment, particularly in America. Judicial interpretation in New York in 1948 established the principle of payment of compensation for partial loss of hearing without loss of earnings. This was the first instance in the compensation field in which payments were made for a partial economic loss to the worker because of the disease. The original basic concept behind workers' compensation was, of course, to provide payments for loss of earnings and for medical costs.

Symons in 1957 emphasized that the proper approach of all parties concerned in this matter (medical, legal, industrial and insurance) should be not how to avoid paying compensation, but rather to accept it as a current problem and try to prevent the need for it where possible; to assess the damage well, and to pay compensation for it adequately if it cannot be prevented.

We have to find out what noise levels in industry are likely to be injurious. We must also know what constitutes a disabling loss of hearing. We must determine the proper method of testing hearing and of arriving at a percentage hearing loss. We must also try to make allowances for the expected drop in everybody's hearing with age. Finally, we must decide what is our criterion for defining "disability" — whether it should be economic loss or social loss of hearing.

The problem affects not only the employee, the employer and the insurance company, but also the public, which in the long run pays the cost of all compensation awards in the price of the manufactured article.

That compensation of industrial deafness is an important problem is realized when one hears that it has been estimated easily to exceed the combined awards for all other types of injury. Moreover, the accrued liability of all industries will be staggering once the principle of paying compensation for industrial deafness is established and recognized by the employee. Symons makes a plea to ignore the social aspects of the deafness and to adhere strictly to the "wage-loss" principle in

assessing these cases. As has been noted earlier, the precedent has already been set to pay compensation even when there has been no loss of earnings, and it is likely that this will continue to apply in the future.

What constitutes hazardous noise?

It is fair to say that no claim for compensation should be considered if the exposure was to a noise of less than 100 decibels' intensity. There are a number of everyday situations, unrelated to work, which carry a noise intensity of just below 100 decibels, and it is not fair to ask industry to carry the responsibility for damage that may have occurred elsewhere. Furthermore, an intensity situation of 100 decibels or more must be endured for more than 90 days before a claim for compensation can be considered.

Before any award should be considered the worker should be removed from the stated source of damaging noise for at least six months, to allow for recovery from auditory fatigue, and thus permit an estimate of the remaining permanent loss of hearing to be made.

How do we assess the hearing loss?

We employ either (*a*) pure-tone audiometry, using the three spech frequencies of 500, 1000 and 2000 cycles, or (*b*) speech audiometry, which requires much more complicated apparatus and yet gives no better results. We use a formula which relates varying percentage values to the degrees of hearing loss. We allow for age presbyacusis by deducting 0·5% per year of age over 50 years from the final percentage obtained. We always express our assessments as "binaural" loss—that is, the percentage below normal hearing with both ears hearing together. Thus, for example, one ear may be totally deaf and the other normal. Our scale in such a case makes the hearing 13% below normal binaural hearing (not 50% as might be thought). In all severe cases an opinion should be stated by the medical examiner as to whether the hearing can be improved with a hearing-aid.

The taking of pre-employment audiograms will sooner or later be an accepted procedure in all heavy industries. We know that some 25% of the community have a hearing loss of some sort anyway, quite unrelated to any noisy work that they may be doing.

Accepting the fact that we shall some time have to deal with hundreds of claims for compensation for deafness arising out of industrial noise, we should make the following procedures a routine in all noisy industries: (i) the taking of pre-employment audiograms; (ii) routine audiometric testing, to try to weed out susceptibles; (iii) noise abatement by engineering and architectural means; (iv) the use of ear protectors (inserts, muffs and helmets), which help because they tend to block the damaging higher frequencies; (v) noise measurement, so that we can know which jobs are potentially dangerous.

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THE PROBLEM OF NOISE IN INDUSTRY.[1]

By W. F. COOPER

Melbourne.

INDUSTRIAL NOISE may be a physical hazard, capable of producing permanent partial deafness. As with other physical hazards, potentially dangerous noise levels must be defined, recognised and eliminated or controlled.

[1] Read at a meeting of the Section of Public Health, Industrial Medicine and Hospital Administration with the Section of Oto-Rhino-Laryngology, Australasian Medical Congress (B.M.A.), Tenth Session, Hobart, March 1 to 7, 1958.

In 1830, in England, Fosbroke first recorded hearing loss in blacksmiths, and later in that century similar deafness was reported in railway men and weavers. Legge and McKelvie in 1927 noted that 24% of 1000 cotton weavers studied had some degree of deafness. All these persons had worked for more than ten years in that industry.

Occupational hearing loss from noise may stem from acoustic trauma by exposures to blasts and explosions, or more commonly by exposure to continued noise at a hazardous level, with the production of nerve deafness. To prevent this, industrial medical departments set up conservation of hearing programmes. The initial advised step is a noise analysis survey of the industry concerned, to define all areas where the noise has reached damaging levels. A damage-risk criterion curve for varying sound intensity and frequency levels is the most important standard to be set up and worked with practically in such a preventive programme, which has the following two main objectives: (i) to conserve the hearing of workers and minimize hearing loss; (ii) to prevent economic loss to the employer resulting from occupational hearing losses.

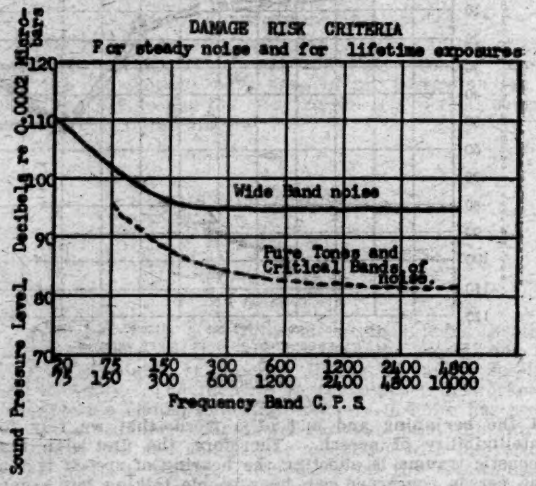

FIGURE I.

Figure I portrays current thinking on this subject. It was orginally suggested in 1953 by Rosenblith and Stevens, two United States Air Force research workers. The vertical scale is in decibels and the horizontal scale in frequency octave bands. If values above this sliding standard of 110 db. in the lower frequencies to 95 db. in the higher frequencies represent sound at the employee's exposed ear level, they will produce deafness in a worker. Values below the curve will not carry a damage risk. This curve is for the average typical industrial noise with its broad range of tones. If, however, the component noises are mainly one critical band, or pure tone, deafness will be produced at a lower level, as shown by the "pure tones and critical bands of noise" curve. The damage is caused by disruption and degeneration of the sensory cells of the inner ear, with secondary degeneration of the ganglion cells.

The five main factors in consideration of a noise's potential to produce deafness are its over-all intensity, its frequency spectrum, the time distribution of exposure to noise during a typical work day and the total duration of noise exposure during expected work life, together with individual susceptibility, for which as yet there is no validated predictive test.

These suggested figures represent the damage risk for a worker's life-time exposure. The adoption of a curve

simplifies the problem, suggesting as it does a thorough inspection of all suspected areas with two instruments, the noise level meter and an octave band analyser, and sometimes oscilloscopes and other electronic apparatus.

A preliminary screening to identify probable hazardous areas may be rapidly carried out by conducting speech level interference observations, and as is shown in Figure II, it will be appreciated that the damage-risk

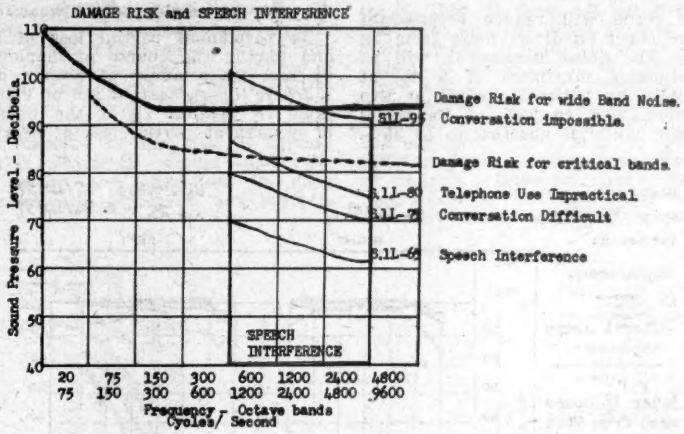

FIGURE II.

criterion curves represent quite high sound intensity levels.

When a noise source is doubled or increased by 100%, the decibels are increased by only three, the decibel scale being a logarithmic one. Conversation must be impossible and telephone use impracticable at the two damage-risk criterion levels suggested. Pain may be

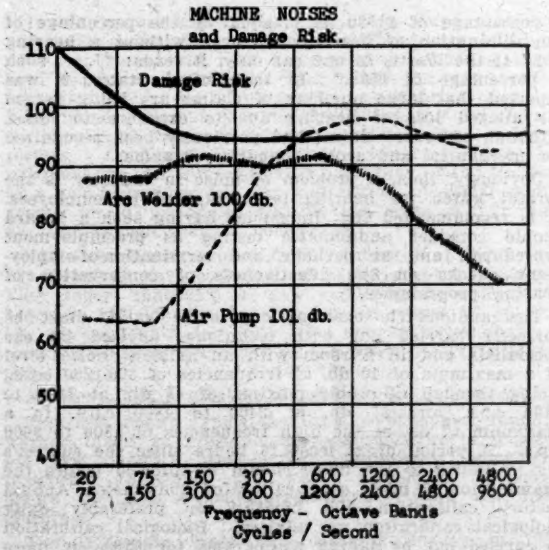

FIGURE III.

experienced at levels of 130 db. We must not forget that both intensity and frequency are necessary determinations, because a simply loud noise may not contain harmful factors.

As is shown in Figure III, we can have two wide-band industrial noises, each in the area of 100 decibels, but only one of them may be capable of producing deafness. This is the air pump, because its frequency content rises above the damage risk criterion curve while the arc-welding noise does not.

When these areas are recognized industrially, it is necessary to eliminate or control the noise with reference to the human ear in these environments. There are some seven main methods of attack, which will now be described.

Vibration Mountings.

Alteration of machine mountings to resilient or rigid supports may be tried to reduce the noise in frequencies of 300 c.p.s and below. High-frequency noise, being emitted directly into the surrounding air, will be unaffected.

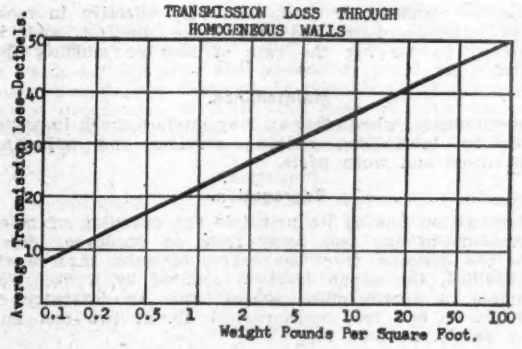

FIGURE IV.

Enclosures.

Enclosures are the most predictable and by far the best method of isolating sound, especially high-frequency noise. Enclosure depends for its attenuation on the physical laws of transmission loss.

Figure IV shows how a non-porous homogeneous barrier attenuates noise by approximately 8 db., where the wall weighs one-tenth of a pound per square inch, to 50 db. for 100 pounds. The suppression is thus four to five pounds each time the weight of the wall is doubled. Increased effectiveness can be gained by splitting the wall and interpolating an air space. Sound will travel

through any crack or fine passage, so that all doors must have an air-tight gasket. Even sealing a cinder block wall with plastic paint will double its attenuating value, but of course reduces its sound absorption on that side. Most efficient transmission loss in the 300 to 9000 cycles per second frequencies can be obtained by acoustic treatment of two pieces of three-eighths of an inch plywood with a two to six inch air space between.

Acoustic Treatment.

Acoustic treatment of walls will reduce reverberant noise. Because it has no effect on direct noise from an operation to an operator, the noise attenuation will be of the order of an estimated maximum of 5 db. at 10 feet from a source with no noticeable effect at two feet. Average walls reflect 90% to 95% of incidental noise, but efficient acoustic material absorbs up to 95%.

Gas-flow Muffling.

Gas-flow muffling is cheap and very effective in most noisy compressed-air and gas-release ducting, and is achieved by slowing the rate of flow or muffling the outlet.

Maintenance.

Maintenance, when efficient, may produce much improvement by lubricating rubbing services and adjusting unbalanced and worn parts.

Segregation.

Segregation has as its principle the grouping of noisy operations in one area away from an employee. Each time the distance from the source of noise in free air is doubled, the sound level is reduced by 6 db. For example, a single noise source with an intensity of 100 db. at one foot measures 94 db. at two feet and only 88 db. at four feet.

Modification of Equipment.

Intelligent change in operations can help in noise reduction plans. Giving a shearing stroke to a punch press reduces its noise, and slowing the speeds of motors and fans reduces the intensity and frequency of the noise they produce.

Other Considerations.

If in any factory areas it is impracticable or uneconomical to abate the noise effectively below the damage-risk criteria, then a barrier must be interposed between the incident sound waves and the employee's ear. This may be done with various ear protective devices, such as plugs of rubber, "Neoprene" or cotton impregnated with wax; incidental noise is reduced by an average of 30 db. Ear muffs and helmets together may produce a possible maximum attenuation of 50 db., but only in the higher frequencies. Plugs must be reseated from time to time through the working day.

Industrially, consideration must be given to the declaration of such noisy areas as places where employees are required to wear ear protection at work, and it is suggested that such areas should be designated wherever a recording of 85 db. and over is noted for average over-all noise. The best ear protection is, of course, the one the employee will wear.

As permanent partial loss of hearing arising out of and during the course of employment is compensatable disease, lump sums of money are claimable. In the *Workers' Compensation Act* of Victoria, 1953, the amended table in Section 11 of the principal Act in the case of permanent partial loss of hearing states: " . . . such

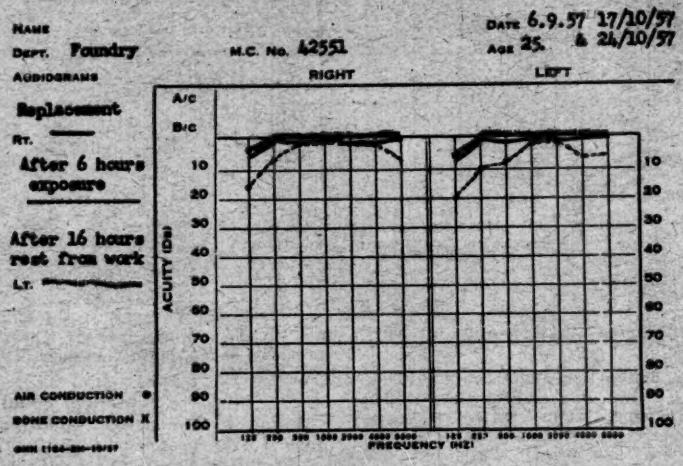

FIGURE V.

a percentage of £1680 as is equal to the percentage of the diminution of hearing measured without a hearing aid." If the loss is in one ear only, it reads: " . . . such a percentage of £560." In the United States, it was reported that large numbers of claims are being lodged for alleged loss of hearing due to exposure to noise, although of course claims had previously been recognized for mechanical and acoustic auditory trauma.

Obviously, then, a problem of noise in industry is the careful watch for hearing loss sustained by employees. It is recommended that industries having such a hazard should consider audiometric testing as preemployment procedures, and as periodic and termination-of-employment checks on the effectiveness of conservation of hearing programmes.

The audiometric examination, to be valid, must be correctly carried out with techniques advised by ear specialists and in a room with an ambient noise level of a maximum of 40 db. at frequencies of 300-1200 c.p.s., rising through allowable maxima of 48 db. at 1200 to 2400 c.p.s. and 57 db. at 2400 to 4800 c.p.s. to a maximum 67 db. at the high frequencies of 4800 to 9600 c.p.s. A period of at least 16 hours since the subject's last exposure to the noise should be allowed before the examination is made on a calibrated audiometer. Annual factory calibration and monthly or preferably daily biological calibration are advised. Biological calibration is carried out by taking audiograms for three or more young workers who have no history of previous ear disease or hearing loss, and who should therefore be within 5 db. of the audiometer zero threshold at all frequencies.

With new employees, the taking of a careful personal and family history and the results of ear, nose and throat findings must be recorded, and decision made

as to whether the employee will be allowed to enter a known noisy environment. Employees with deviations from normal should be referred to an ear, nose and throat specialist for diagnosis, particularly in cases in which his opinion is required to separate the losses of hearing due to disease or presbycusis from hearing loss induced by a previous occupation.

It is certain that conductive deafness is no barrier to an employee's entering such an area, being equivalent to "built-in" ear plugs; but there are two schools of thought about perceptive or neural deafness, with the theory on one hand that as such persons have already suffered nerve damage, their initial susceptibility has been lost. Of course, as Sataloff advises, the problems of recruitment and the cause and degree of the perceptive deafness must be considered. Kuhn is of the opinion that new employees presenting with established neural deafness should not be exposed to the risk of further damage. The industrial physician should seek the consultant otologist's advice and direction in such questionable matters in selective placement and periodic evaluation.

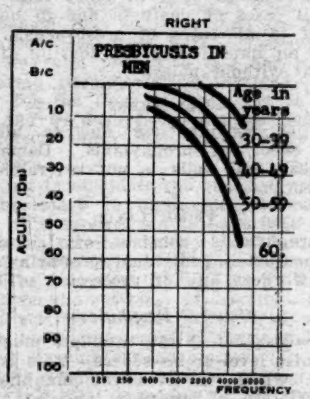

FIGURE VI.

It is recommended that at least two consistent pure-tone audiograms be made independently with all new employees, and that an audiogram showing threshold hearing be established each month for two months, and then every six months when no hearing loss has been noted. Should the employee's hearing deteriorate, he should be induced to wear more effective ear protectors. Continued deterioration would indicate his reference to an ear specialist, together with transfer to a non-hazardous job and the taking of serial audiograms for a period of six months, after which time final assessment may be considered.

The Sub-committee on Noise in Industry of the American Academy have collected much statistical information. They report that 25% of new employees have impaired hearing; 15% have a hearing loss in excess of 20 db.; 5% of applicants for jobs who are examined have a severe high-tone loss; 10% of the losses are in one ear only.

Auditory fatigue must be allowed for in the audiograms.

Figure V shows typical audiograms of a new foundry worker taken at preemployment examination and after his first month's work. The audiograms show the temporary threshold shift which completely recovered before the man recommenced foundry work the next morning.

The Wisconsin State Fair Hearing Survey in 1954 gave interesting presbycusis curves in men, showing the effects of age for four decades.

Figure VII demonstrates the "4000 dip" in the threshold curve which is typical of repeated exposure *to damaging noise. Data from the Sub-committee on* Noise previously referred to indicate that losses occur at frequencies above those that characterize exposure sounds. It is most important to protect the speech range of frequencies from 500 to 2000 c.p.s., so we must pay major attention to the 300 to 600 c.p.s. and 600 to 1200 c.p.s. bands as the most potentially hazardous from a long-range viewpoint, with 85 db. and above as the potentially dangerous levels in these bands.

It is recommended that any decibel loss in excess of 15 at any frequency be regarded with suspicion as possibly indicative of a noise-induced hearing loss. According to Kuhn, unless noise has an alarm factor in it, then it does not adversely affect production or efficiency, and ear plugs in a noisy environment actually allow speech communication to be more easily effected. This is probably due to the reduction of over-all masking noise and the more critical octave components of the consonants and vowel speech sounds. An affected employee typically does not subjectively realize his hearing loss, which may be only in the higher voice registers and in musical appreciation. He may complain of tinnitus. However, should no conservation-of-hearing programme be instituted, this may in severe cases, by continued nerve degeneration, cause a loss spreading to

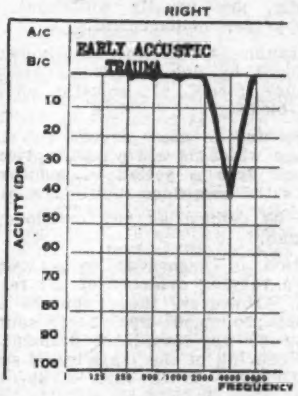

FIGURE VII.

the higher frequencies and restrict an employee's domestic and social life. Difficulty may be encountered in transfer to other types of occupation, where efficient conversational appreciation may be essential.

When an employee has become deafened by repeated noise damage, the condition is incurable, and a rehabilitation programme to make the most of his remaining hearing and to educate him in the boundaries of his auditory appreciation must be undertaken.

Conclusion.

In conclusion, then, effective conservation-of-hearing practices to prevent problems stemming from industrial noise are based on four steps: determination of noise levels and frequencies; reduction of excessive noise at its source; the use of protective devices by employees; a comprehensive audiometric programme.

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THE PROBLEM OF NOISE IN INDUSTRY.[1]

By NEIL REILLY, M.B., B.S., D.L.O., F.R.A.C.S.
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OCCUPATIONAL HEARING LOSS is a term used to denote impairment of hearing which arises in, during the course of or as the result of one's employment. The loss may be partial or complete, and it may affect one or both ears. It may be classified under the following causes: (i) mechanical trauma, (ii) acoustic trauma, (iii) noise exposure.

Mechanical trauma may result from blows to the head as well as from local trauma to the region of the outer ear, ear canal or ear-drums. It may follow the introduction of foreign bodies or result from burns of the ear-canal or ear-drum. Injury to the ear may be followed by perforation of the drum or infection of the middle ear, and is occasionally associated with inner-ear involvement. The resulting hearing loss can be of conductive, perceptive or mixed type. These hearing impairments occur suddenly, are usually unilateral and tend to recover with proper management.

Acoustic trauma refers to the immediate loss of hearing produced by one or several exposures to very sudden, intense forms of acoustic energy, such as explosions or blasts.

Hearing loss due to noise exposure is the cumulative loss of hearing which is always perceptive in type, and which develops over a period of months or years in employment with hazardous noise levels.

Noise may be defined as any over-loud, excessive or unwanted sound.

The definition of hazardous noise levels is difficult because of the varying evidence of the research workers in this field. However, there appears to be general agreement that prolonged exposure to sounds of greater intensity than 80 db. results in permanent damage to the high-tone hearing of the majority of subjects exposed to the sound. That is to say, 80 db. is the critical intensity level.

It is not overall sound intensity that matters most. Intensities at the various octave band levels may vary greatly in the composition of the overall sound level. The damage produced has regard for the frequency of the damaging tone, with the greatest effect at the 4000 cycles per second level in the cochlea.

SYMPTOMS.

When first exposed to intense noise many workers find it distressing, but after a few days or weeks they appear to become used to it. It is not for some years that subjective symptoms become evident, because this type of deafness commences in the high tones, and the important speech frequencies (500, 1000 and 2000 c.p.s.) are preserved until severe damage has occurred.

Buzzing, or ringing tinnitus occurs in about 50% of cases, and other subjective sound phenomena may occur —for instance, Larsen's patient, who reported that at night he had "an exact imitation of the dockyard noise with all its different sounds in the ears".

Vertigo is uncommon, and it has been suggested that, in those cases in which it occurs, it is due to exposure to strong vibrations at the same time as exposure to damaging noise.

SIGNS.

In the typical case clinical examination reveals no abnormality, but audiometric testing shows a dip at 4000 c.p.s.

In the early stages the dip may extend only over the two octaves, 2000 to 8000 c.p.s., with its lowest point at about 30 db. Gradually the dip widens and deepens, and ultimately the audiogram comes to approximate closely to that seen in cases of presbyacusis.

Speech audiometric testing shows the articulation curve to be almost identical with that seen in presbyacusis.

DIAGNOSIS.

The typical subject is a healthy, young to middle-aged man, without history or evidence of ear disease, whose history does not suggest injury by intoxication or some severe constitutional disease or deafness in the family, but reveals prolonged exposure to noise, and whose audiogram shows a high-tone loss of the type referred to above.

The 4000 c.p.s. dip is important for diagnosis in the early stages, but is not pathognomonic for deafness due to exposure to noise. It may occur in intoxications due to tobacco, quinine, cocaine or alcohol, in syphilis, in head injury, in otosclerosis and in retinitis pigmentosa. In the later stages the differential diagnosis from presbyacusis may be difficult.

It is worthwhile to keep in mind that regression is present in all cases of deafness due to noise exposure, but is absent in all cases of presbyacusis. That is to say, there is an improvement of hearing after the lapse of an interval without noise.

TREATMENT.

Treatment of hearing loss due to noise exposure consists, in effect, of prophylaxis. Damage sustained by the cochlear hair cells is not reversible and, therefore, is untreatable.

PROPHYLAXIS.

General prophylaxis consists of technical and of organizing measures. Individual prophylaxis consists of selection of workers and of protection of the ears.

General Prophylaxis.

Technical measures.—First, attempts must be made to reduce the noise level at its source. This is an engineering problem, which involves the manufacture of the machines, variation of processes and so on. Secondly, attempts must be made to prevent the noise spreading from its source. This is a mixed engineering and architectural problem, which involves positioning of the machines, their isolation, sound-proofing and sound absorption in the buildings, and so on.

Organizing Measures.—First, if possible, the workers should be given the opportunity of changing to a less noisy process from time to time. Secondly, if possible all breaks and rest periods should be spent in quiet surroundings. These general measures cannot prevent the occurrence of deafness, but they may delay the onset and decrease the degree of permanent cochlear damage.

Individual Prophylaxis.

Selection of Workers.—Complete otological and audiological examination of all workers before employment in noisy occupations is essential. It is generally agreed that workers with an existing hearing loss due to cochlear disease or to otosclerosis and those with a predisposition to hearing loss should not be employed. There is some disagreement about those with an existing hearing loss due to middle ear disease, although it would appear that this type of trouble acts in a protective fashion so far as hearing loss from noise exposure is concerned. It has the effect of raising the critical intensity level.

Although there is an apparent raising of the critical intensity level in hearing loss due to cochlear lesions when tests are made with a fatiguing tone, the inability of the hair cells to respond owing to their diseased condition makes a greatly increased intensity necessary to produce an apparent fatiguing effect. This greatly increased intensity then has a further damaging effect on the already diseased hair cells. That is to say, the apparently raised critical intensity level not only gives a false picture, but also carries with it the possibility

[1] Read at a meeting of the Section of Oto-Rhino-Laryngology with the Section of Public Health, Industrial Medicine and Rehabilitation, Australasian Medical Congress (B.M.A.), Tenth Session, Hobart, March 1 to 7, 1958.

of unsuspectingly exposing an ear to a sound level which will cause increased damage.

After much investigation and research into the problem, the Sub-committee on Noise in Industry of the Committee on Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology recommends that air-conduction preplacement audiograms should be prepared for all workers at least at the frequencies of 500, 1000, 2000, 3000, 4000 and 6000 c.p.s. The sub-committee goes on to make the following statement:

> If a preplacement audiogram shows an average hearing loss of more than 15 decibels at 500, 1000 and 2000 cycles per second, the employee should be referred to a physician for otological examination and placement evaluation before assignment to a job. Such referral is also advised for employees whose audiograms show any unusual irregularity, particularly an abrupt loss beginning at 2000 cycles per second. Whenever preplacement tests are given, the tester should ascertain that a period of at least sixteen hours has elapsed since the subject's last noise-exposure.

Follow-up tests of the hearing of workers exposed to noise above the critical intensity level are essential. It is well known that some persons are highly susceptible to hearing loss from noise exposure, but up till the present no valid predictive test has been elaborated by which these people can be detected. Therefore it is necessary for the first follow-up test to be performed between 60 and 90 days from commencement of employment, to detect those who show early signs of a 4000 c.p.s. loss.

In attempting to elucidate the question of susceptibility, it is first necessary to distinguish between adaptation and fatigue; this appears not to have been done in much of the literature on this subject. We must go back to Adrian's definitions, which are as follows: Adaptation means a decline in excitability caused by the stimulation, quite apart from the existence of activity. Fatigue means a decline in activity caused by previous activity of the organ — that is, a difference in physiological character. An effect due to auditory adaptation (i) is greatest at the stimulating frequency, with a symmetrical rise and fall on each side, (ii) is nearly of the same size, whatever frequency is used (that is, there is no frequency specificity), (iii) is never accompanied by tinnitus, and (iv) as a rule is of shorter duration than the stimulation. An effect due to auditory fatigue (i) is greatest half an octave above the stimulating frequency —that is, it is asymmetrical in relation to the stimulating frequency, (ii) is greatest at 4000 c.p.s. (that is, there is frequency specificity), (iii) is often accompanied by tinnitus, and (iv) as a rule is of longer duration than the stimulation. Out of this, from the practical point of view, comes the necessity for a 16 hours' interval between the last noise exposure and testing. So testing is best done at the beginning of a shift, for preference after a night's sleep. This, in its turn, raises the question of practicability. It is manifestly impossible to take complete pure-tone audiograms of all workers on a shift just prior to beginning work. To shorten the time of testing many schemes have been tried, and some form of screen testing must be used. For the first follow-up test a screen test at 15 decibels' loss should be carried out at all the six recommended test frequencies. If a worker's test shows a shift of more than 15 db. at any one of these frequencies, he should be submitted to full audiological assessment. If the screen test shows no loss greater than 15 db., it should be sufficient to carry out further tests at intervals of 12 months.

The first screen test should be performed earlier than 90 days after employment, (i) if the worker is exposed continuously to noise levels greater than 100 db., (ii) if he complains of excessive discomfort or tinnitus, or (iii) if he complains of excessive temporary hearing loss.

Later follow-up tests may be confined to measurements of the 4000 c.p.s. frequency, provided that the worker's loss of hearing at 4000 c.p.s. is not greater than 50 db. It has been found that within the 50-decibel limitation, the loss at 4000 c.p.s. is as much as or more than the loss at any of the other so-called "recommended" frequencies. Therefore, the value of a threshold shift at 4000 c.p.s. may be considered an upper limit for any threshold shift that may have occurred at any of the other frequencies. When a single frequency screening test is used, it is wise to test at at least two levels 15 db. apart, to detect changes of the order of 15 db. It has been shown that if the loss at 4000 c.p.s. is greater than 50 db., it is not an adequate measure of the loss at other frequencies. Therefore, if the worker's loss at 4000 c.p.s. is greater than 50 db., a threshold test at all frequencies must be performed. A single-frequency test is of great value, because large numbers of workers can be tested in a short time, it may be performed in any reasonably quiet room and it does not require highly trained personnel for its performance.

If a large change is found in the hearing of a worker, the question of his reassignment to other work in the factory or of his discharge from employment must be considered. Before this is done, at least three threshold audiograms should be prepared on different days prior to any noise exposure and at least 16 hours after the last noise exposure.

Protection of the Ears.

A very great deal of work has been done in an attempt to arrive at some form of local ear protector which will prevent the damaging effects of prolonged exposure to noise, at the same time permitting adequate communication by speech with neighbouring workers. A large number of ear plugs and ear muffs have been devised. Some have selective attenuation affecting the frequencies from 1000 c.p.s. upwards. Most cause a more or less even attenuaton over the whole range. In these the average attenuation is about 20 to 25 db., so that they will not protect the cochlea from damage by noise intensities of over 100 db.

Whatever form of ear plug is considered advisable and supplied by an employer as part of a hearing conservation programme, the greatest difficulty lies in inducing the worker to wear the plugs. They seem to prefer to risk permanent damage to their hearing than to wear plugs and risk ridicule by some of their workmates.

Implementation of Prophylaxis.

Much has been written into arbitration Acts and factories Acts and agreements between unions and employers concerning protection in hazardous occupations, but it seems that little has been done to protect the worker from the effects of prolonged exposure to noise. There may be provisions in some of the Acts and agreements in some of the States, but there is no general statement anywhere of the real necessities in this regard.

Hazardous noise levels can be found in parts of most factories, some more so than others. It should be a legal obligation on the part of every employer not only to supply an adequate number of efficient ear defenders for his employees, but also to carry out the technical and organizing measures referred to above, as well as pre-employment and follow-up audiometry. Failure to carry out these obligations should be punishable.

Similarly there should be a legal obligation upon every employee to adopt the means of protection offered him by his employer, and failure to do this should also be punishable.

Factories should be inspected by officers appointed under the particular Act applicable. These officers should be adequately trained in audiology in its broadest sense. They should use sound-level meters with octave-band analysers to map the noise levels of the whole factory, as well as of the individual places of working of the employees. They should be able to advise both employer and employee, and they should be empowered to report to the proper authority cases of failure to carry out the obligations referred to above.

It is only in this way that it will be possible to hope to achieve alleviation of this very real hazard.

JET NOISE.

So far I have discussed what may be called the ordinary industrial noises; but we must not forget that nowadays, and more so in the future, jet noises are coming into the picture and are bringing with them their own peculiar problems.

Jet noises take us outside the immediate area of the factory. People are involved in the factory, including the testing areas; in the airport in checking, testing and warming up the engines; on the airport as personnel involved in other duties, who nevertheless are exposed to the noise of jet aircraft frequently and repeatedly; on the airport as passengers and visitors; and in areas neighbouring the airport as residents.

The problems of jet noise are many, ranging from simple annoyance at a distance, to masking of normal means of communication closer to the source and, most important, to possible hearing loss amongst those working quite near to the source of the noise.

Here it is not simply a matter of exposing man to a noise of a different nature. The intensity has been increased greatly above that at all common in factories, greater than the levels at which audiological scientists have been used to working. In the case of jet aircraft with afterburners the noise levels exceed 140 db. in many instances, while we must remember that the threshold for pain is about 135 db.

Many of the problems arising in connexion with noise of this intensity are discussed in "An Exploratory Study of the Biological Effects of Noise", published by the University of Chicago, in which the subject is examined by many experts. In addition to the more obvious sections dealing with noise levels and ear protection, evidence is produced that jet noise can directly affect the vestibular system by exceeding the threshold of 135 db. in the middle frequencies. Exposure to more than 140 db. appears to produce some postural difficulty, apparently unassociated with the vestibular system. It is suggested that there may be a direct effect on the reticular formation giving rise to central nervous system symptoms, in particular changes in some deep tendon reflexes. It is possible that these very intense sound levels may give rise to a marginal stress syndrome with complaints of tiredness, irritability, insomnia and loss of libido.

It is obvious that the investigation of these effects of high-level noise calls for the employment of many whose fields of interest have lain outside the auditory system, as well as of all those who are employed in investigating the various angles of the problem of exposure to ordinary noise. The answer also involves architectural and engineering attacks upon the source of the noise in the engine, the provision of work bays to reduce spread of noise, the screening effects of trees and buildings in preventing the escape of noise from runways, and other related problems.

Here, again, it would seem likely that the degree of final protection achieved will require the imposition of legal obligations, this time on the authority responsible for the siting of airports, as well as on employer and employee.

EVALUATING THE DISABILITY OF HEARING LOSS.

Hallowell Davis has made the following statement:

> It is not easy to construct a set of rules for translating hearing loss into dollars to be paid as industrial compensation. Nevertheless such rules are needed to take care of present cases of industrial hearing loss and also as a yardstick for any criteria that are ever to be drawn up as to what is a "dangerous" or a "permissible" noise in industrial situations.
>
> There are four distinct steps between the hearing loss and the dollars of compensation. One is the actual measuring of the hearing loss, usually with an audiometer; another is the conversion of the measurements into a single figure, usually a percentage of some sort; the third is the setting of a dollar value for one hundred per cent disability and specification of the conditions under which payment should be made for a noise-induced hearing loss; and the fourth is the establishment of a "causal relation" between the particular hearing loss and the noise that is presumed to have caused it.
>
> The amount of compensation to be paid for total disability of hearing is not a medical or technical matter. It must be fixed by law, by court decisions and industrial commissions. It is here that the question of wage loss as a condition for compensation must be settled.
>
> The method of measuring hearing loss and the rules for arriving at a "percentage of disability" must also be approved by legislation, but here scientific as well as social and economic factors are involved. As yet there is no general agreement on methods of measurement and rules for calculations.

In *The Journal of the American Medical Association* of April 16, 1955, an article was published by the Council on Physical Medicine and Rehabilitation of the American Medical Association, approved by the Committee on Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology, in which were set out the various principles which should be considered in evaluating hearing loss. The Council pointed out first that the method recommended by the Council on Physical Medicine of the American Medical Association in 1947 was fairly accurate for conductive losses but not for cochlear lesions, and secondly, that there was no present means of converting the hearing loss as shown by pure-tone audiometry accurately into actual loss for speech. Also, the conventional spoken-voice and whisper tests were inaccurate, and covered only a narrow range of intensity. It was still necessary to standardize methods of use and rules for the interpretation of speech audiometric findings, but it agreed that the basis for evaluating hearing loss must be that of everyday communication without the patients' being given any artificial aid or any special training. This should be so because of the wide acceptance of the principle that a loss of normal function should be evaluated in the absence of any prosthetic device.

The Council defined hearing for speech as "the ability to identify spoken words or sentences under average everyday conditions of normal living by the sense of hearing, unaided by touch or vision", and stated that in evaluation it was the function of hearing for speech that was important, not some anatomical injury.

It was further pointed out that it was not yet possible to define precisely what were the limits of normal hearing, or what comprised total inability to hear speech, but that "the limit of normal for hearing of speech 'might' reasonably be assumed for the present to be comparable to that for the hearing of pure tones in the speech frequency range". It was necessary to evaluate the loss in each ear separately, as well as arriving at a value for binaural hearing. Allowances should be made for psychological factors and for malingering, as well as for the patient's educational standard. Finally, a clinical diagnosis had to be reached, and as part of the evidence for this both air-conduction and bone-conduction pure-tone audiograms should be taken.

CONCLUSION.

If we in Australia hope to be able to achieve some standards for evaluating hearing loss, we must bear in mind the work of the American Medical Association, and look forward to the time when further research on its part will establish more accurate methods than are now available. In the meantime, we should set up a standards committee which can start to work on this problem as it affects this country, and as our very first step we should standardize the calibration of our audiometers to give us some base line from which to operate. This should be able to be achieved easily, because the Commonwealth Acoustic Laboratory has adopted the American standard, and each of us may have our audiometers regularly and frequently calibrated by means of the 6 c.cm. coupler which is available at the Acoustic Laboratories in the various States.

The next need is for a standard audiogram, which, if possible, should be of an internationally agreed design.

It is not necessary for me to stress the lack of opportunity for research work in Australia in audiology in general, as well as in the particular field discussed in this paper. It could well be one of the functions of the committee which I have suggested to investigate the possibilities of research in Australia and, if possible, to start something in this direction.

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THIACETAZONE AND THE ERYTHROCYTE SEDIMENTATION RATE IN TUBERCULOSIS.

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THE erythrocyte sedimentation rate, almost since its estimation was introduced, has been regarded as one of the most important criteria of activity of the tuberculous process. Lately, however, the validity of this assumption has been questioned on several grounds. Only recently W. R. Horsfall, of Cairns, showed that there were racial differences in North Queensland which affect the response of the sedimentation rate to tuberculosis (Horsfall, 1957). Similarly H. Malluche (1950) and others found that the sedimentation rate was altered by thiacetazone treatment of tuberculous patients. Thiacetazone (4-acetylaminobenzaldehyde-thiosemicarbazone) is one of the tuberculostatics that have been introduced in the last 15 years, and is also used in the treatment of Hansen's disease. The present studies[1] were concerned with the background of the slowing process in the sedimentation rate (estimated by the Westergren method) brought about by thiacetazone.

Experimental Investigation.

The substance used in the experiments was water-soluble thiacetazone supplied by the manufacturer,[2] and the blood was from patients with advanced active tuberculosis. A 3·8% solution of sodium citrate was used for the Westergren estimation of the erythrocyte sedimentation rate, and for the addition of thiacetazone the same pipette (producing a drop of approximately 0·05 ml.) was used in all the experiments. The blood was taken from the cubital vein of the patients at approximately 9 a.m.

Experiment I.—An amount of 3·2 ml. of blood was added to 0·8 ml. of citrate solution, and the mixture was divided into two portions, each of 2 ml. To one of them, one drop of 10% thiacetazone solution (2·5 mg. of thiacetazone per millilitre of citrated blood) was added, and the sedimentation rate was read after one hour and two hours. The results are set out in Table I.

TABLE I.

Tube Number.	Subject.	Erythrocyte Sedimentation Rate (Westergren) (Mm.).		Diagnosis.
		Without Thiacetazone.	With Thiacetazone.	
1	Pa.	2/3	1/2	No abnormality.
2	Sc. (1)	42/71	5/10	Tuberculous spondylitis with fistula formation.
3	Mo.	56/95	2/6	Progressive pulmonary tuberculosis with cavitation.
4	Mn.	68/89	1/2	Generalized skeletal tuberculosis with fistula formation.
5	Sc. (2)	37/68	2/3	Tuberculous spondylitis.
6	Sc. (1)	45/75	1/2	Tuberculous spondylitis with fistula formation.
7	Fr.	101/121	2/5	Progressive pulmonary tuberculosis.
9	Sc. (3)	38/60	1/2	Tuberculous spondylitis with hypertension.
11	Sc. (4)	83/102	7/15	Progressive pulmonary tuberculosis.
12	Sp.	77/100	3/5	Progressive pulmonary tuberculosis.

[1] In Table I and subsequent tables the first figure represents the value at the end of the first hour and the second figure (after the downstroke) the value at the end of the second hour.

Experiment II.—In this experiment, 8 ml. of blood were added to 2 ml. of citrate solution, and the mixture was divided into five portions of 2 ml. each. The first portion served as control. To the next was added one drop of 10% thiacetazone solution, to the third one drop of 10% PAS solution, to the fourth one drop of 10% streptomycin solution, and to the fifth one drop containing 250,000 units of penicillin G. All the tubes were put on to the stand

TABLE II.

Erythrocyte Sedimentation Rate (Millimetres).

Tube Number.	Subject.	Control.	With Thiacetone.	With PAS.	With Streptomycin.	With Penicillin.
1	Mn.	68/98	1/2	53/85	47/68	56/90
2	Ve.	Failed	2/2	30/51	20/41	25/46
3	Sc.	37/63	2/3	41/62	35/56	32/60
4	Sc.	45/75	1/2	45/70	40/75	37/70
5	Fr.	101/121	2/5	11/115	95/122	92/121
6	Pl.	65/108	6/22	92/118	53/94	59/100

[1] The experiments were carried out in 1947-1948 at the *Landes-Lungenheilstätte Marburg/Lahn;* the senior staff physician was the late Dr. E. Wulff.

[2] "Solvoteben" (Bayer, Leverkusen, Western Germany); the drug was developed there by Domagk and co-workers (1948).

immediately, and the results were read after one hour and two hours. The results are set out in Table II.

Experiment III.—In the third experiment, 16 ml. of blood were added to 4 ml. of citrate solution, and the mixture was divided into 10 equal portions. The first portion served as a control. To the second was added one drop of 10% thiacetazone solution, to the third one drop of 1% solution, to the fourth one drop of 0·1% solution, and so on. The results are set out in Table III.

TABLE III.

Tube Number.	Erythrocyte Sedimentation Rate. (Millimetres.)	
	First Hour.	Second Hour.
1	56	95
2	2	6
3	62	92
4	64	94
5	59	88
6	56	86
7	60	87
8	59	90
9	54	87
10	57	91

Experiment IV.—In this experiment, 9·6 ml. of blood from each of two patients were added to 2·4 ml. of citrate solution and the mixture was divided into five equal portions. The first served as a control. To the second was added one drop of a 2% solution of thiacetazone, to the third one drop of a 4% solution, and so on. The tubes were put up on the stand immediately, and hourly readings were made for 12 hours. The results are set out in Table IV.

TABLE IV.

Erythrocyte Sedimentation Rates (Millimetres) of Two Patients.

Subject.	Hour.	Control.	Concentration of Thiacetazone.				
			2%	4%	6%	8%	10%
Patient A.	1	83	75	60	25	9	7
	2	102	97	90	65	20	16
	3	115	110	101	85	30	24
	4	119	117	113	109	45	34
	5	120	118	114	111	60	45
	6	122	118	115	115	72	58
	7	123	120	116	117	80	61
	8	124	120	117	117	85	69
	9	124	120	118	119	102	80
	10	124	124	124	126	116	97
	12	124	124	125	126	120	101
Patient B.	1	77	52	50	43	18	3
	2	100	95	88	87	49	5
	3	106	108	114	98	87	10
	4	108	111	117	102	97	26
	5	109	113	119	103	100	45
	6	112	118	120	104	102	59
	7	112	118	120	104	102	67
	8	112	118	120	104	102	70
	9	112	120	120	104	102	72
	10	112	120	120	104	102	76
	11	112	120	120	104	103	81
	12	112	121	121	104	105	81

Experiment V.—In the fifth experiment, 32 ml. of blood were added to 8 ml. of citrate solution, and the mixture was divided into 10 pairs of portions, each of 2 ml. The first portion of each pair served as control. To the second portion of the first pair a drop of 10% thiacetazone solution was added immediately, and to the second portions of subsequent pairs the drop of solution was added consecutively at intervals of one hour, just before they were put up into the stands. The blood came from one patient. The results are shown in Table V.

Experiment VI.—In the sixth experiment, 9·6 ml. of blood were added to 2·4 ml. of citrate solution, and the mixture was divided into two pairs of portions each of 2 ml. The first 2 ml. of each pair served as control. To the second 2 ml. of each pair, one drop of 7% thiacetazone solution was added just prior to the placing of the tubes in the stands. The first pair was put up immediately, the second pair after 24 hours and the third pair after 48 hours. The results are set out in Table VI.

Experiment VII.—In the seventh experiment, 24 ml. of blood were taken and added to 6 ml. of citrate solution. The mixture was divided into 15 portions each of 2 ml. The first five (Group I) served as controls; to each of the second five (Group II) one drop of thiacetazone solution was added at zero hour; to each of the third five (Group III) a drop of thiacetazone solution was added immediately before the tubes were put up in the stand. The first portions of each group were put up on the stand immediately; the second and subsequent portions were shaken and put up at intervals of one hour. The results are set out in Table VII. The dilutions of thiacetazone solution were always freshly prepared from 10% solution in ampoules, immediately prior to being added to the citrated blood.

TABLE V.

Sedimentation Rate (Millimetres) Read at One Hour and Two Hours After Tubes had been Put up in Stands.

Time of Addition of Thiacetazone Solution (Hours).	10% Thiacetazone Solution Added.		Control.	
	1 Hour.	2 Hours.	1 Hour.	2 Hours.
0	1	2	38	60
1	1	1	30	52
2	2	5	29	56
3	1	3	31	60
4	2	4	33	62
5	1	2	39	70
6	3	5	42	68
7	1	2	39	76
8	0·5	2	38	75
9	1	2	37	74
10	2	2	36	74

Experiment VIII.—In the eighth experiment, 3·2 ml. of blood were added to 0·8 ml. of citrate solution, and the mixture was divided into two portions. To one portion one drop of 6% thiacetazone solution was added; the other served as control. Tuberculous patients whose erythrocyte sedimentation rates estimated by the Westergren method were known to be the same were used. The results are set out in Table VIII. Previous tests had shown that the addition of one drop of physiological saline to 2 ml. of citrated blood did not influence the erythrocyte sedimentation rate to any particular extent, and therefore it was not added to the controls.

TABLE VI.

Sedimentation Rate (Millimetres) at One and Two Hours in Experiment VI.

Time of Putting Up. (Hours.)	7% Thiacetazone Solution Added.	Control.
0	20	33
24	17	31
48	110	94

Clinical Investigation.

The change in the erythrocyte sedimentation rate during six weeks' treatment with 50 to 100 mg. per day of thiacetazone given orally to 53 tuberculous patients was compared with that in 291 tuberculous patients who received treatment with PAS or streptomycin, or non-specific therapy. The comparability of the subjects was established on bacteriological, radiological and clinical criteria. Both thiacetazone-treated patients and controls had all given at least one positive bacteriological finding, and both radiologically and clinically the general comparability of the groups was satisfactory. The results are set out in Table IX.

Conclusions.

The following conclusions were reached as a result of the investigation:

1. The addition of 0·05 ml. of 10% aqueous solution of thiacetazone to 2 ml. of citrated blood (2·5 mg. of

thiacetazone per millilitre of citrated blood) substantially reduces the erythrocyte sedimentation rate (Westergren) of tuberculous as well as of healthy subjects (Experiment I).

2. The addition of the same amount of PAS or streptomycin, or of 250,000 units of penicillin G, to 2 ml. of citrated blood does not produce this effect (Experiment II).

TABLE VII.

Erythrocyte Sedimentation Rate (Millimetres) in Experiment VII.

Subject.	Concentration of Thiacetazone.	Group.	Age of the Citrated Blood. (Hours.)				
			0	1	2	3	4
First Hour Values.							
St.	0	I	34	33	38	37	39
Ga.	0	I	26	31	28	24	18
Ma.	0	I	36	36	36	38	40
St.	7%	II	12	16	17	19	19
Ga.	7%	II	8	27	29	24	19
Ma.	8%	II	6	8	9	14	41
St.	7%	III	10	7	5	3	2
Ga.	7%	III	6	3	3	3	2
Ma.	8%	III	7	3	3	3	1
Second Hour Values.							
St.	0	I	61	62	67	63	70
Ga.	0	I	49	60	56	49	37
Ma.	0	I	67	67	68	69	74
St.	7%	II	26	34	35	40	43
Ga.	7%	II	21	58	56	49	43
Ma.	8%	II	17	20	26	35	77
St.	7%	III	22	16	11	6	4
Ga.	7%	III	11	5	6	5	4
Ma.	8%	III	17	8	9	7	5

3. The borderline concentration of thiacetazone necessary to produce this effect with certainty lies at about 1 mg. per millilitre of citrated blood (Experiments III and IV).

4. The slowing effect of 2·5 mg. per millilitre of thiacetazone in citrated blood disappears when the blood is left standing for more than 12 hours (Experiment IV).

TABLE VIII.

Erythrocyte Sedimentation Rate (Millimetres).

Subject.	Before Addition of Thiacetazone.		After Addition of 6% Thiacetazone Solution.	
	1 Hour.	2 Hours.	1 Hour.	2 Hours.
Neu.	53	85	7	16
Ker.	50	90	3	6
Gra.	57	95	7	16
Geb.	54	89	2	5
Adl.	53	80	20	40
Ros.	58	100	9	23
Kri.	55	85	18	40

5. The disappearance of the slowing effect depends on the concentration of thiacetazone in the standing citrated blood; the higher the concentration, the later the disappearance of the slowing effect (Experiment IV).

6. The slowing effect does not diminish, but increases, when the citrated blood is left standing up to 10 hours, provided that the thiacetazone is not added to the standing blood until immediately before it is placed in the stands (Experiments V and VII).

7. Even when a borderline concentration of fresh thiacetazone solution is added to old citrated blood, the slowing effect may still be noticeable after 24 hours (Experiment VI).

8. The slowing effect of a borderline concentration of thiacetazone on the citrated blood of patients with active tuberculosis is variable, and is not related to the erythrocyte sedimentation rate (Experiment VIII).

9. The behaviour of the erythrocyte sedimentation rate of 53 tuberculous patients, treated with 50 to 100 mg. of thiacetazone per day for at least six weeks, did not differ significantly from that of 291 controls treated with streptomycin or PAS or by non-specific methods.

Discussion.

As the therapeutic dosage of thiacetazone, well tolerated over many weeks, does not substantially exceed 100 mg. per day given by mouth, it is not surprising that the braking effect appears only occasionally, and does not affect the average figure. About 0·5 to 1 mg. of the drug is administered per pound of body weight. Therefore, the therapeutic plasma concentration should as a rule lie below the slowing threshold of the drug.

TABLE IX.

Comparison of Changes in Erythrocyte Sedimentation Rate of 53 Patients Treated with Thiacetazone and 291 Patients Treated Otherwise (Six Weeks).

Erythrocyte Sedimentation Rate.	Thiacetazone Group. (53 Patients.)	Control Group. (291 Patients.)
Unchanged normal	5	20
Improved	30	149
Stationary (±10 mm.)	7	61
Deteriorated	11	61

That water-soluble thiacetazone slows the erythrocyte sedimentation rate, and that this slowing effect depends on time and concentration, have previously been shown by Malluche (1950), and his findings are confirmed here. However, Malluche assumed that a change in the plasma must have been responsible for the disappearance in the slowing effect. Certainly the plasma changes on standing as serum. From Experiments IV to VII in the present paper, however, it appears that changes in the plasma cannot be responsible for the disappearance of the slowing effect. If this had been so, the slowing effect would have disappeared whether the thiacetazone remained in the standing citrated blood or was freshly added later. The only possible conclusion is that thiacetazone is unstable in dilutions below 10%, just as it is unstable in aqueous solutions below 10% for storage purposes. A diminution in its therapeutic effect is to be expected as a result of its disintegration.

Therapeutic Rationale.

1. When thiacetazone is administered to tuberculous patients and immediate slowing of the erythrocyte sedimentation rate takes place, this is good evidence that therapeutic concentrations of the drug are present in the plasma.

2. The in-vitro results suggest that frequent administration of thiacetazone may produce higher effective concentrations in the plasma than less frequent administration of larger doses, because the dilution of the drug in the plasma must be high.

Summary.

Experimental evidence confirms the fact that thiacetazone in its water-soluble form slows the erythrocyte sedimentation rate (Westergren) in healthy and tuberculous subjects, and that this slowing effect diminishes with time and with concentration of thiacetazone in citrated blood.

It has been shown that this diminution of the effect does not take place, but that slowing is increased, when thiacetazone is added freshly to citrated blood which has been left standing for four and even 24 hours. It has also been shown that the same concentrations of PAS and streptomycin, as well as penicillin, do not produce the slowing effect, and that tuberculous patients with similar sedimentation rates do not show similar degrees

of slowing of the rates. No average difference in the changes in the sedimentation rate was observed between 53 tuberculous patients (after they had received 50 to 100 mg. of thiacetazone per day by mouth for six weeks) and 291 controls.

From these experiments, I conclude that therapeutic plasma levels of thiacetazone do not often reach the slowing threshold, because of the dilution of the drug and its concomitant instability. If slowing occurs, I believe it to be good evidence that therapeutic levels have been achieved. More frequent administration may produced higher effective levels than less frequent administration of larger doses.

Acknowledgements.

I am indebted to the Department of Pharmacology, University of Sydney, for information; to my patients, who agreed without hesitation to allow extra blood to be taken from their veins; to my wife, who diligently helped with the estimation of the sedimentation rates, and to the firm of Bayer, Leverkusen, who supplied the water-soluble thiacetazone free of charge.

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Reports of Cases.

MAMMARY NEOPLASM IN A MALE RECEIVING ŒSTROGEN THERAPY FOR CARCINOMA OF THE PROSTATE.

By JOHN E. MOULTON, M.B., B.S.,

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THE difficulty in making an accurate diagnosis of a lump in the breast of a male has increased since the introduction of œstrogen therapy for the relief of the symptoms arising from carcinoma of the prostate. A case is presented in which this difficulty arose, and the methods for ensuring a more accurate diagnosis are discussed.

Clinical Record.

The patient, a man aged 80 years, was initially admitted to the Repatriation General Hospital, Concord, in 1953, with increasing difficulty of micturition, diurnal and nocturnal frequency, poor stream and scalding. A rectal examination was carried out and a large, nodular, stony-hard prostate was discovered. The chest, lumbo-sacral bodies, spine and pelvis were radiologically normal. The blood urea content was 44 mg. per 100 ml. and the serum acid phosphatase content was 3·5 units per 100 ml. (normal, 1 to 3 units per 100 ml.). A diagnosis of carcinoma of the prostate was made, and treatment was commenced with stilbœstrol, in a dosage of 5 mg. three times a day for 21 days, then 1 mg. three times a day for the rest of his life. The response to this drug was dramatic, and the patient was completely symptom-free for three years. He was admitted in September, 1956, to another hospital, complaining of further urinary difficulty, for which a transurethral resection was carried out. Two weeks later a bilateral subcapsular orchidectomy was performed, and the patient was discharged from hospital on an increased dose of stilbœstrol—10 mg. three times a day. Prostatic fragments were examined microscopically, and carcinomatous tissue was found in each piece examined.

On January 18, 1958, he was readmitted to the Repatriation General Hospital, Concord, suffering from hæmaturia, and rectal examination revealed that the prostatic growth had greatly increased in size and had almost blocked the rectum. The serum acid phosphatase content, on this admission, was 2·5 units per 100 ml., the serum alkaline phosphatase content was 12 units per 100 ml. (normal, 5 to 10 units per 100 ml.) and the fasting blood urea content was 100 mg. per 100 ml. The hæmaturia settled quickly after rest, and the patient was able to pass urine without difficulty. The dosage of stilbœstrol was reduced to 1 mg. three times a day and the patient appeared very comfortable, when, 10 days after his admission, a large, tender, hard and irregular mass, involving and occupying the entire left breast tissue, suddenly appeared. The mass, measuring 8 cm. by 3 cm. by 4 cm., was attached to the skin, but not to the underlying muscle; the nipple was retracted and surrounded by an eczematous area. The right breast was also enlarged, but was soft and flabby and contained several mobile, non-tender lumps. Hard but mobile glands were felt in the left axilla, while the right axilla was clear. X-ray films of the pelvis and ribs revealed doubtful areas of metastatic deposits. Closer questioning of the patient revealed that both breasts had become enlarged and tender six months previously, but that the stilbœstrol dosage had not been reduced. After two weeks the tenderness disappeared, but, whereas the right breast returned to normal, small lumps could still be felt in the left.

Stilbœstrol was suspended and a diagnosis of left mammary carcinoma secondary to prolonged and massive stilbœstrol therapy (15,950 mg. approximately) was made. On February 14 a simple mastectomy was carried out, and although the patient appeared to be doing well immediately after operation, the combination of age and neoplastic disease caused a rapid regression in his condition, and he died on March 13.

A full post-mortem examination was carried out, and secondary deposits of malignant tissue were found in both the right and the left breasts, and in the liver, the lungs, the abdominal lymph nodes (particularly the aortic group), the right and left axillary lymph nodes, the peritoneal cavity and, by direct extension, the rectum. The microscopic picture of these secondary deposits was identical with that of the primary prostatic growth—namely, a poorly differentiated adenocarcinoma. The pituitary gland showed an area of cystic degeneration with hyperplasia of basophil and acidophil cells. The adrenal glands were normal, both macroscopically and microscopically. Both kidneys were hydronephrotic, with associated chronic pyelonephritis.

Comment.

The importance of this case rests in the fact that, without a full post-mortem examination and histological comparison between the primary prostatic neoplasm and its secondary deposits, a correct diagnosis would not have been made. Clinically, this patient presented with a primary carcinoma of the left breast secondary to stilbœstrol therapy for the relief of carcinoma of the prostate. Since the introduction by Huggins, in 1941, of hormonal therapy for the treatment of prostatic neoplasms, the known side effect of gynæcomastia has been well tabulated. However, it was not until 1948 that Abramson and Warshawsky reported a case of carcinoma in a male breast secondary to œstrogen therapy. The fact that administration of œstrogens may cause malignant changes in the breasts of male mice had been proved by Lacassagne (Nathanson, 1944), and the same influences were thought to apply to the breast neoplasms in this reported case. Since 1948, further cases of hormonally induced malignant disease of the breast have been reported, and concern was felt that stilbœstrol, "wonder drug" though it may be for the relief of symptoms of prostatic carcinoma, was, in fact, a potentially dangerous and carcinogenic drug. However, in a recent paper by Benson (1957), the problem of differentiating between primary and secondary carcinoma developing in the breasts of patients receiving œstrogens is fully discussed, and it is now held that the majority of these so-called hormonally induced primary neoplasms were, in fact, metastatic deposits. These latest findings agree with the work of Foote and Stewart (1945), who believe that the hyperplasia associated with gynæcomastia is not related to neoplastic changes. Moore *et alii* (1945), after careful breast biopsies

in males receiving œstrogens, also doubt that these hormones would cause malignant change. Benson believes that a careful autopsy must be carried out on patients who have died from carcinoma of the prostate, to whom œstrogens had been given and in whom lumps have appeared in the breasts. In most cases other metastatic deposits are found, and in these cases proof of independent carcinoma must be considered lacking. For difficult cases, the breast tissue and prostatic tissue should be analysed for acid phosphatase content. Acid phosphatase studies may give positive results in prostatic carcinoma and should give negative results in primary breast carcinoma. If these investigations are carried out, Benson believes that the diagnosis of a primary breast tumour will be made with less frequency. The apparently real increase in the incidence of metastases to the breasts of hormonally treated patients may be related to the changes occurring in breast tissue during or after such treatment. The œstrogenic substances may, by increasing the vascularity and the amount of loose periductal fibrous tissue, provide a medium in which metastatic tumour cells can be more numerous and can grow more readily. Corbett and Abrams (1950) believe that the increase may be due to the prolongation of the natural history of the prostatic carcinoma afforded by the administration of œstrogens.

The experience of McClure and Higgins (1951) is quoted by Benson as an example of the necessity for post-mortem examination. They originally reported the case of a patient who developed bilateral breast carcinomata after receiving œstrogens for carcinoma of the bladder. The findings obtained from biopsies of breast and bladder tissue were interpreted as distinct histologically, and a diagnosis was made of primary carcinoma of the breast secondary to œstrogenic therapy. However, at later autopsy, careful study revealed that the carcinomatous tissues in both areas were indistinguishable, the breast tumour being, in fact, metastatic.

To McClure and Higgins's case can be added the case presented here, for without autopsy this also would have been labelled a hormonally induced primary breast neoplasm.

Summary.

1. The incidence of metastatic deposits in the breasts of patients with carcinoma of the prostate receiving œstrogen therapy is increasing or being increasingly recognized.

2. Primary hormonally induced breast neoplasia is still extremely rare.

3. The need for post-mortem and enzymatic studies is emphasized before the diagnosis of an independent breast tumour is made in such cases.

Acknowledgements.

I wish to thank the Chairman of the Repatriation Commission for permission to present this case, Dr. T. J. Constance for his histological reports and advice, and Dr. A. L. Carrodus, Dr. R. M. Dunn and Dr. J. Blackwood for their interest and help.

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Reviews.

Neurotic Distortion of the Creative Process. By Lawrence S. Kubie, M.D.; 1958. Lawrence: University of Kansas Press. 8¼" x 5", pp. 160, with illustrations. Price: $3.00.

THIS book will appeal to those concerned with creativity in education, art, science, finance and other fields of endeavour. The content, as Kubie's bibliography indicates, is the distillate of much clear thought and penetrating insight. The book is small, attractive and easy to read, an example of the creative process allied with literary craft. Lucid and arresting prose matches apt diagrams clearly explained. In enticing sequence each proposition is stated, elaborated, then summarized; each is self-contained, but prepares for the next, permitting pause at any point. General conclusions are reached from various starting points and in various contexts, building up conviction, Pelion upon Ossa. We have but a few small criticisms.

In Chapter I, "The Psychodynamics of Neurosis and Creativity" Kubie states three purposes: (i) to show the universality of creativity and the neurotic process; (ii) to reexamine the belief that links genius with insanity, creativity with illness; (iii) to discuss the noxious assumption that one must be ill to be creative.

In plain language and diagram, a reliable formulation of relevant basic psychoanalytical theory leads to the definition: "A creative person is one who has retained a freer use of his preconscious functions." Creativity, essentially a function of the preconscious, may be restricted or distorted by the rigidity of conscious functions, or the stereotyped repetitiveness of unconscious conflicts. Examples are drawn from dreams, art, literature, scientific research and finance. The implications for education, including medical education and research, are tremendous.

Chapter 3, "Education for Preconscious Freedom and its Relation to Creativity and to the Process of Maturation", postulates that we need not, and cannot, be taught to think; we need to learn how not to interfere with this inherent capacity of the mind, and how to acquire the tools of communication. Educational procedures that fail to recognize that creative thinking is continually braked and diverted by unconscious forces perpetrate a fraud. Reason and example are marshalled to support this, especially the folly of erudition without wisdom or maturity.

Chapter 4, "General Summary", shows the edifice built thoughtfully upon the foundations of the early chapters, a classical exposition and application of psychoanalytical concepts and insights, open to challenge, but commanding respect, provoking thought and pointing to an educational reform.

Anxiety in Christian Experience. By Wayne E. Oates; 1958. London: George Allen and Unwin, Limited. 8¼" x 5¼", pp. 160. Price: 15s. (English).

THIS is a book about normal anxiety. Specifically, it deals with the kinds of anxiety experienced by every Christian in his pilgrimage. These are economic anxiety, finitude anxiety, the anxiety of grief, the anxiety of sin, legalistic anxiety, the anxiety of the Cross, holy dread and the fellowship of concern. The book also discusses the anxiety reactions of the morally indifferent.

It is a worthy successor to the author's earlier book, "Religious Factors in Mental Illness", which was favourably reviewed in this Journal on April 20, 1957, (page 552). Oates is Professor of Psychology of Religion and Pastoral Care at Southern Baptist Theological Seminary, U.S.A., and he has had extensive experience as a pastoral counsellor, and as a chaplain in mental hospitals and psychiatric clinics. What he has to offer in this volume should be interesting and helpful to all doctors and their patients, as well as to ancillary medical personnel and clergymen. For instance, it falls to the lot of most doctors to try to offer some word of comfort and encouragement to the relatives when a patient dies. Many find the task difficult and embarrassing; but it is likely to become easier and more effective if, in future, it is carried out in the light of Oates's chapter on the anxiety of grief, with its excellent description of the various emotional stages of the grief process.

From the psychiatric point of view the author's approach is eclectic, and this book deals mainly with anxiety at conscious levels, though the subconscious is not ignored. Two criticisms may be mentioned. In the chapter on the

"morally indifferent", he assumes that these people, who are often psychopaths, can profit by experience. It would be better to differentiate between true psychopaths, who are unable to learn from experience, and the morally indifferent, who have the capacity, but who have not had the opportunity, to learn moral principles. Secondly, in the concluding chapters, which deal more directly with the anxiety of man's personal relationship with his Maker, he says little about redemption.

The last chapter is a noble exhortation to be "genuinely anxious" for others, and will strike a responsive chord in the hearts of all good doctors.

The Physical Treatment of Varicose Ulcers: A Practical Manual for the Physiotherapist and Nurse. By R. Rowden Foote, F.I.C.S., M.R.C.S., L.R.C.P., D.R.C.O.G.; 1958. Edinburgh and London: E. and S. Livingstone, Limited. 7¼" × 4¾", pp. 135, with 86 illustrations. Price: 15s. (English).

This is an excellent little book which, while written ostensibly about the physical treatment of leg ulcers, contains much concentrated wisdom on the whole subject of varicose conditions, expressed in the simplest manner. It is a book to be studied and not just read, for a wealth of detail is packed into it. So we find in the preface two basic truths: "Physiotherapy compression treatment and simple exercises will heal and keep healed the vast majority of ulcers"; and "Whenever possible sticky compression bandages should be applied by the surgeon in charge of the clinic". Later, in Chapter 1, in dealing with the importance of the varicose ulcer in the life of the community, the author makes the following statements:

(1) All hospitals should have a specialised department for the treatment of varicose disease and ulceration.

(2) These departments should be under a surgeon prepared to give the necessary time to the specialty.

(3) Continuity of administration as far as a surgical appointment is concerned is essential.

The book is divided into two parts. The first covers the importance of the ulcer problem, the anatomy and physiology of the venous system, common types of ulcers and the general principles of treatment. This section is written clearly and gives an easily understandable account of all the factors underlying the principles of treatment. The second part gives a detailed account of the methods of treatment, bandaging, massage, physical exercises, after-care and specialized electrical forms of physiotherapy. This latter section consists of a chapter by Miss Truda Wareham, Superintendent-Physiotherapist, St. Bartholomew's Hospital, London. The importance of after-care is rightly stressed, and a short chapter is devoted to it. It should not be thought that this book advocates physiotherapy as the sole therapeutic measure; its place in treatment is clearly stressed, as is also the correct time for its use. Surgical treatment is mentioned but, naturally in a book of this type, only briefly.

The illustrations and skiagrams throughout are excellent. In the first part they are very well chosen, and blend admirably with the clarity of the writing. In the second part, they are very numerous and detailed, and leave no room for misunderstanding as to the methods of bandaging, massage and exercises. The book is printed on glossy paper and is of convenient size for pocket or handbag. Such a book has long been needed, and should be in every physiotherapy department, in the hands of every tutor sister and of every medical practitioner who undertakes treatment of these leg conditions.

Studies on Fertility: Including Papers Read at the Conference of the Society for the Study of Fertility, Exeter, 1957, being Volume IX of the Proceedings of the Society. Edited by R. G. Harrison, M.A., D.M.; 1958. Oxford: Blackwell Scientific Publications. 8½" × 5¼", pp. 132, with many illustrations. Price: 25s.

The British Society for the Study of Fertility has published Volume IX in its series; this contains selected papers read at the proceedings of that society during the 1957 meeting at Exeter. This volume contains the first Oliver Bird lecture, which has for its purpose the advancement of knowledge on fertility control and contraception. This address deals with the biochemical characters of spermicides and was delivered by Dr. Mann, who has done much research work on the subject. The rest of the volume contains papers relating to original research work on human beings and experimental animals, and thus is spread over a field that extends from gynecology and obstetrics to genetics and veterinary physiology. The book is further subdivided into sections dealing with the male and the female.

The veterinary field is well covered by such papers as that dealing with crystallization patterns of sodium chloride in bovine cervical mucus and their implications in the diagnosis of pregnancy in cows. Other papers discuss genetically determined sterility in the mouse, the maturation of the reproductive organs in the cat, and artificial insemination in pigs.

Readers of this Journal will be more interested in such subjects as the therapeutic value of tubal patency tests compared with that of other procedures, the clinical management of suspected pelvic tuberculosis, the role of suspension operations in infertility, and spermatogenesis following operative cure of undescended testes in the male.

This publication does not cover the usually wide field of human infertility to the same degree as one finds in the proceedings of its counterpart societies elsewhere; but the investigator beset by the problems of human sterility will be repaid by a study of the relevant papers on that subject in this book. It is of interest that the publication of such a volume has been financially assisted by a well-known drug house. The book is handsomely bound, the paper is of good quality and the publishers are to be congratulated.

Alfred Hospital Clinical Reports, Volume 8; 1958. Melbourne: The Alfred Hospital. 9¾" × 5¼", pp. 114, with 10 illustrations and 15 tables. Price not stated.

This volume opens with an appreciation by L. B. Cox of the late J. F. Mackeddie, a prominent and colourful figure in Melbourne medical circles between the wars, who played a leading role in the affairs of the Alfred Hospital during those decades, and is specially remembered for his work in connexion with the establishment of the Baker Medical Research Institute. There follows a collection of reports on various aspects of the work at this hospital. P. J. Parsons reviews the case reports of patients with hæmatemesis and melæna admitted to the Alfred Hospital during the decade 1948-1956, and J. C. Stewart discusses the question of surgical intervention in these cases, in the light of experience during the last three years. H. A. Luke discusses angiography of the aorta and great vessels, with special reference to Seldinger's technique of arterial catheterization, illustrated by an excellent series of radiographs and accompanying diagrams. J. A. McLean gives a report on direct blood transfusion to ambulant patients, prefaced by the statement that 1500 such transfusions have been given at the Alfred Hospital during the past decade. E. A. Alcock contributes a survey of the management of epithelioma of the lips and R. A. Hayes a review of 219 cases of leukæmia observed at the Alfred Hospital during the years 1946-1957. D. G. Duffy describes a case of the Leriche syndrome with hypertension, and W. H. Kitchen two cases of quadriplegia in the new-born. Finally, G. R. Stirling discusses the surgical anatomy of the heart, with special reference to some aspects which seem of particular importance to the conduct of intracardiac procedures under direct vision. The net result is an interesting symposium of some of the work being done at one of our leading hospitals, presented in a manner befitting its source.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Cell and Tissue Culture", by John Paul, M.B., Ch.B., Ph.D., M.R.C.P. Ed.; 1959. Edinburgh and London: E. & S. Livingstone, Limited. 8¾" × 5¼", pp. 272, with 40 illustrations and 16 tables. Price: 30s. (English).

An attempt to provide an up-to-date account of the techniques and applications of tissue culture.

"New Biology", edited by M. L. Johnson, Michael Abercrombie and G. E. Fogg; 1959, No. 28. 7" × 4¾", pp. 138, with illustrations. Price: 4s.

Contains articles on the voyage of the *Beagle*, the structure of Darwinism, the formation of animal species, the origin of isolation, automatic mechanical self-reproduction, and hair.

The Medical Journal of Australia

SATURDAY, MAY 23, 1959.

CIVIL DEFENCE.

> It is vital to understand that in a situation of nuclear equality, between two sides, each of which has the most modern means of delivery, that side will survive which has the best organised home front. The crucial problem will be the defence of home morale—not only the attack on enemy morale. Home morale is the business of Civil Defence. Civil Defence is vital to the modern fighting chief, because without it his base is not secure.
>
> —FIELD MARSHAL VISCOUNT MONTGOMERY.

CIVIL DEFENCE is, perhaps more than anything else, a medical problem—or at any rate, the sort of problem with which medical people are familiar, and in which they are involved at least as much and as vitally as anyone else. This of course presupposes that we are thinking of civil defence in its currently accepted sense—all that is bound up in Viscount Montgomery's reference to "the defence of home morale"—and not as some form of civilian armed force. Indeed, the present conception of civil defence is by no means limited to, though it certainly includes, the circumstances of war. It has been credited with two main aims: (i) mitigation of the effects of enemy attack by saving life and maintaining morale, and (ii) alleviation of distress caused by natural disaster. In parts of Australia a civil defence organization is already functioning in terms of the second aim and has played a valuable part in face of the devastation caused by floods. Such an organization is thoroughly worth while in itself and should be encouraged in every way for its immediate value to the community, but it is also a sound basis on which to build for civil defence in the event of enemy attack, especially attack with atomic weapons.

Is such an attack possible or likely? Likelihood is something the ordinary citizen cannot readily assess. It must largely be left to our leaders, who have more information and better advice at their command than most of us, and we can only hope that the present lack of a positive government policy on the matter is well based. As to possibility there can be no doubt. It seems clear that a major atomic weapon could be landed on any Australian city with little warning, perhaps virtually none, and cause great devastation. The optimum power of such a weapon is considered likely to be of the order of ten million tons of T.N.T.—usually referred to as ten megatons. Exploded at ground level this might be expected to cause by its blast effect alone total destruction for a distance of four miles, irreparable damage for seven miles and moderate to severe damage for thirteen miles. Heat would kill all who were caught in the open up to four miles and cause third degree burns to about thirteen miles, second degree burns to about fifteen miles and first degree burns to about twenty miles; in addition, it would ignite combustible materials up to a distance of thirteen miles. Immediate nuclear radiation, in the form of gamma rays, would be a danger for about a minute; the distance at which there would be a dose giving a 50% chance of survival in the open would be about two and a quarter miles, but building materials etc. would considerably reduce the dose by shielding. The danger from residual radiation would lie chiefly in the gamma rays given off by fall-out and might occur close to and (depending on meteorological conditions) at a great distance from a nuclear explosion. This last-mentioned part of the total hazard has been much stressed in popular writing and impresses by its insidiousness, but it is in some respects the part least to be feared if adequate protective measures are adopted.

It will be evident from this very brief account of the effects of the explosion of a single ten-megaton weapon that very great damage would be done. Nevertheless, the danger can be exaggerated and over-dramatized, so that paralysing despair results. This is a false approach, as despite the magnitude of the problem it should be possible to do a great deal to meet an attack. Even in Great Britain with its concentrated population and small over-all distances, the problem is being faced realistically. Its essential aspects have been outlined by Sir Ernest Rock-Carling and G. R. Stanbury,[1] who conclude their article as follows:

> Notwithstanding the appalling difficulty, every Authority has faced up to the situation: food and feeding; blood and blood product supplies; water supplies; transport; communications; monitoring; evacuation of people and of resources have all been planned. When the worst possible picture has been presented to the responsible authorities it has seemed possible in every case to give a service, at least in the less affected areas.
>
> There are grounds upon which a defeatist attitude to thermonuclear bombing can reasonably be assumed but it should be made clear that they do not include radioactivity unless there is total absence of preparation.

In the United States and Canada with their large centres of population and heavy industry, but with greater distances, a vigorous programme of civil defence is being pursued. The authorities are making a determined attempt—evidenced, for example, in a published interview[2] with Leo A. Hoegh, Director of the Office of Civil and Defence Mobilization in the United States—to inform the general public of the hazard and of how it can be met by the ordinary citizen as an individual and as a unit in civil defence. The medical profession has been alerted, and that not recently if we note that the Ninth County Medical Societies Civil Defence Conference was held in Chicago in November, 1958. The sixteen papers[3] presented cover a wide range of the subject. This annual conference was sponsored by the Council on National Defense of the American Medical Association and is "planned to inform and otherwise assist other health personnel for their respective roles in disasters".

[1] *Practitioner*, 1958, 181: 598 (November).
[2] *Today's Health, 1959, 37: 36 (January).*
[3] *J. Amer. med. Ass.*, 1959, 169: 348 (January 24).

Whether or not too much is being made of possible atomic attack in the United States it would not be profitable to discuss here. More important to us is whether too little is being made of it in Australia. A few people have thought a great deal about it, and some sound planning has been done. Such a study as that by David Storey on the expansion of a civil hospital which we published recently[1] is to be commended. The Commonwealth Civil Defence School at Macedon in Victoria has undoubtedly enlightened many people and, while presenting grim realities, has shown that the situation is not without hope. From a medical point of view there is much to be thought about, and there is no doubt that the medical profession would spring to the challenge if a policy were enunciated. Such a policy is needed. Even if it is thought on the best advice that the possibilities of an atomic attack are quite remote, nothing would be lost by officially approving and promulgating a plan for the basic organization. Key people, such as local government authorities, doctors and others, should be given something specific to think about and an opportunity to work out their own possible contributions to an over-all scheme. A further statement from Field Marshal Viscount Montgomery sums up the position:

> The future will call for a civil defence organization which exists in peace to the degree necessary to ensure it can operate in top gear in any emergency. It must be understood in this respect that while great destruction may be caused at the point of burst of a nuclear weapon, tremendous saving of life and property will be possible on the fringe . . . Civil defence must be moved up to take its rightful place in the national war machine.

Current Comment.

THE INSANITY OF MAN.

HUGH WOODWORTH, in a recent book,[2] has put forward an intriguing thesis that every individual man and woman is insane. Whilst many writers have had fleeting impressions of a similar type, they have hesitated to put them in print. This author is confident, and backs his theory by reference to life and evolution. The insanity began with primitive man. The advent of human thinking created an ability to remember and review memories. At a stride man was superior to the whole animal kingdom; but a price had to be paid. Animals could live with fear limited to the immediate present; man was doomed to a myriad fears of his own invention. His life had the daily imprint of a thousand great or small fear-provoking associations, for which memory was responsible.

Man has a tremendous drive to security, Woodworth tells us. This embraces the fourfold elements of material security, social security (i.e., the approval of society), emotional security and intellectual security. Man must look ahead year by year to provide for the emergencies of living—children, old age, even burial. He is literally "afraid" of what people think of him, his clothes, his connexions. If he has the prize of matrimony, he possesses a fragile "her" which may be lost. Every possession carries with it a fear of losing it. Desperately he clings to religion, politics and philosophy; but despite them "the world is split into terrified armed camps by a difference in ideology". Cravings and anxieties dog his footsteps. Importantly, even worthwhile objectives—a university degree or a home—need years of effort and planning.

> They thus naturally give rise to thousands of sub-objectives, steps on the way, each of which becomes charged with an anxiety of its own. We are afraid we may not pass the exam, we may not make the sale, we may not arrive on time.

Woodworth asks us to think out our own problems seriously and to draw our conclusions. Are we addicted to compulsive emotional thinking tinged with anxieties? This situation is certainly ubiquitous. It is stupid and harmful. Ought we to face reality and use the term insane?

The reader will wonder if there is an escape from this universal psychosis. The remedy lies in the practice of unemotional thinking. The situations which produce fear must be closely studied and understood. Escape by emotional subterfuge must be eliminated. Anger, fear, anxiety disappear when questions are squarely faced. The approach is similar to that of the business man who said: "I have no worries, I have merely problems." In addition, we must relearn the arts of childhood, to enjoy our pleasures with rapture and without the intrusion of self. This is the moral based on common sense of "Sanity, Unheard Of". It makes stimulating reading.

THE HARMFUL EFFECTS OF EXCESS INSULIN.

THE estimation of the daily doses of insulin necessary for the treatment of diabetes is still largely empirical. Early attempts were made to estimate the requirement on a rational basis by assuming that the amount of insulin required could be calculated on a stoicheiometric basis from the extent of the hyperglycæmia and of the glycosuria. Attempts were made to determine the ratio G/I (G = grammes of glucose utilized, I = units of insulin injected). Different authors put out different figures from less than one to over twenty. Further, in any patient the ratio was not the same from day to day or from hour to hour with the same dose of insulin. M. Somogyi and H. V. Goldwasser[1] have now studied the results published by authors over the past 30 years as well as their own results, and they show that the magnitude of the G/I ratio is largely dependent on the quantitative relationship between the amount of carbohydrate ingested and the amount of insulin injected, which can be expressed as a ratio R. In every case presented, the G/I ratio increased when R increased and became less when R decreased. There was, however, wide variability, and G/I did not parallel R.

In a following paper M. Somogyi[2] studies this finding, attempts to explain its causation and discusses the exacerbation of diabetes by excess insulin action in relation to his hypothesis. Over some years he studied in great detail several patients, who had what appeared to be unmanageable diabetes, and arrived at some interesting conclusions, as well as converting the "unmanageable" cases into cases of relatively mild diabetes. The first distinct pattern which emerged from a close study of the data was that there were periodic fluctuations in the glycosuria and hyperglycæmia, which occurred at more or less regular intervals. The high glycosuria tides tended to follow days when the sugar in one or two urine fractions was close to zero. From these results it appears that excessive glycosuria (hyperglycæmia) is an aftermath of hypoglycæmia, and indeed that there is a cause-and-effect relationship between hypoglycæmia and the ensuing glycosuria. A patient given a certain dose of insulin shows increasing glycosuria and hyperglycæmia. The dose of insulin is increased, and, after an interval of hypoglycæmia, even very slight, there is still further increase in the glycosuria, and the condition may progress to very acute diabetes, with ketonuria. By reducing the

[1] Med. J. Aust., 1959, 1: 649 (May 16).

[2] "Sanity, Unheard Of", by Hugh Woodworth, 1958. Victoria, British Columbia: The Sumas Publishing Company Limited. 8½" x 5", pp. 112. Price: $3.00.

[1] *Amer. J. Med.*, 1959, 28: 165 (February).

[2] *Amer. J. Med.*, 1959, 28: 169 (February).

intake of insulin gradually, Somogyi has shown that when much less insulin is given, the patient keeps in equilibrium with little or, in some urine specimens, no glycosuria. The causal connexion between excess insulin action and exacerbation of the diabetic syndrome is very clearly shown in the protocols of experiments given by Somogyi. Physiological studies, published from 1924 to the present day, supply unequivocal evidence to the effect that insulin hypoglycæmia stimulates the adrenal-pituitary system to an accelerated release of blood sugar raising hormones. When the blood sugar level falls to a "critical" level, there is an increase in the rate of adrenaline secretion. This excites the anterior pituitary lobe to an increased secretion of ACTH, and this in turn leads to increased adrenocortical activity. This train of substances acts as insulin antagonistic factors, which overcome the effect of insulin—hence the hyperglycæmia in spite of raised insulin concentration. Curiously, all the data published on this subject had little impact on clinicians. It is evident that in insulin therapy, avoidance of hypoglycæmia, even of mild, asymptomatic degrees, is no less important than the control of excessive hyperglycæmia and glycosuria. By carefully studying the insulin requirements and giving the minimum amount necessary to keep the patient in equilibrium, even if this means some glycosuria, we may prevent the development of a severe state of diabetes and restore patients with severe diabetes to a status of mild diabetes that can be managed with small doses of insulin, or, not infrequently, without insulin. Substantial reduction in the insulin dosage is not advisable and may even be dangerous. The changes should be made gradually with frequent laboratory checks.

In a third paper Somogyi[1] discusses the diabetogenic effect of organic hyperinsulinism in patients with islet cell tumours of the pancreas. Many of these patients show a marked deterioration in response to the carbohydrate tolerance test, develop severe hyperglycæmia and have been diagnosed as diabetics. Somogyi suggests that these phenomena are the result of the diabetogenic action of hypoglycæmia induced by the excess insulin.

The injection of insulin into non-diabetic persons for reducing body weight or for shock treatment in cases of mental disorder can bring about disturbances in the carbohydrate metabolism which are virtually identical with those in "organic" hyperinsulinism: the blood sugar shows diurnal fluctuations between extreme hyperglycæmic and extreme hypoglycæmic levels, and glucose tolerance tests performed during the treatment give results characteristic of diabetes. In such cases the patient is suffering from artificial hyperinsulinism and shows the results of the induced hypoglycæmia. It would seem that careful consideration of the results obtained by Somogyi in the treatment of diabetes would ease the lot of many diabetics.

A SHORT HISTORY OF ANATOMY FROM THE GREEKS TO HARVEY.

It is more than thirty years since Dr. Charles Singer first published the material taken from his Fitzpatrick Lectures in a small book entitled "The Evolution of Anatomy". At that time it was hailed as a masterpiece of scholarly research on a subject which hitherto had received little attention as a separate study among the writings of competent medical historians. For many years this book has been out of print. In 1957 Dr. Singer revised and added to the original edition, which reappears[2] under the new title "A Short History of Anatomy from the Greeks to Harvey".

The opening chapter contains an interesting account of the earliest attempts of the Greeks to break away from the ancient State and religious systems which had completely dominated and controlled the minds and lives of men for long ages. Greek philosophers became critical of the traditional beliefs and ideologies, and in a free spirit of inquiry sought to understand the actual workings of Nature, using inductive methods in observation and research. From the sixth century B.C., new knowledge was acquired in the fields of medicine and biology at the schools of Sicily, Ionia and Cos, where the dissection of animals was carried out and much thought was given to the phenomena of generation. In the course of time many of these advances were recorded in various treatises belonging to the Hippocratic Collection.

The Greek period of scientific investigation ends with the remarkable biological studies of Aristotle, the philosopher-naturalist, and Theophrastus, the botanist, who exercised their personal influence in the establishment of the Alexandrian School, where, aided by comparative anatomy and human dissection, the anatomical descriptions of Herophilus and the physiological studies of Erasistratus brought lasting fame to this Egyptian institution for the high quality of its teaching and research. The accumulated knowledge of the Greeks was summarized and placed on a solid foundation in the voluminous writings of Claudius Galen, whose dogmatic pronouncements on the science of medicine and his original exposition of a complete and satisfying physiological system continued to hold sway right up to the time of William Harvey.

After a short account of the mediæval anatomists and the gradual adoption of a regular curriculum in the teaching of the subject at the different European universities, the final and most important section relates to the revival of learning, which encouraged a realistic study of Nature by the Italian artists, physicians and scholars led by Leonardo da Vinci, Andreas Vesalius, Fallopius, Eustachius, Realdus Columbus and others. It is worthy of note here that the earliest comparative anatomist of modern times, the Dutchman, Volcher Coiter, is given his rightful place as a leader in the new approach to embryology, as he paved the way for Fabricius and Harvey in this dynamic field of biology.

Dr. Singer's "Short History" touches on all the basic trends in the evolution of anatomy which eventually formed a secure foundation for scientific medicine and surgery. It is sure to be even more widely read and appreciated than its less mature predecessor.

THE MEDICAL DIRECTORY OF AUSTRALIA: A HASTY REASSURANCE.

The latest monthly newsletter of an Australian Branch of the B.M.A. contains this note:

> The collection of data for the Medical Directory of Australia, 1950 [*sic*], is nearing completion.
>
> Members who have not yet checked and returned their data cards are asked to do so as soon as possible.

Lest we be thought ungrateful for this free publicity for the Directory, given at our request, we draw a veil over the identity of the Branch concerned. However we hasten to reassure everyone interested that the Directory is not really so out of date as this reference might suggest. It may lag a little here and there, although we do our best to overcome this; but it really has advanced beyond 1950. The point is that it is desired to complete the gathering of personal entries for the next edition by the middle of this year. We should like to have all cards returned by June 1, 1959; any doctor who thinks that he has been missed only needs to ask and another card will be sent. The matter is now urgent. It takes many months to collate and print a volume so large and complex as the "Medical Directory of Australia"; and the publication year is intended to be 1960.

[1] *Amer. J. Med.*, 1959, 28: 192 (February).

[2] "A Short History of Anatomy from the Greeks to Harvey", by Charles Singer; 1957. New York: Dover Publications, Inc. 8" x 5", pp. 226, with many illustrations. Price: $1.75.

Abstracts from Medical Literature.

NEUROLOGY AND PSYCHIATRY.

Medical, Psychiatric, and Legal Aspects of Premenstrual Tension.

I. N. PERR (*Amer. J. Psychiat.*, September, 1958) discusses various aspects of the problem of the premenstrual tension syndrome from medical, psychiatric and legal viewpoints. He states that from a review of the literature it appears that the vast majority of women have various elements of this syndrome, but that to establish a diagnosis of premenstrual tension Dalton's criteria should be used, namely, (i) the symptoms must be present in each of the three previous cycles; (ii) symptoms must be severe enough to demand medical advice; and (iii) symptoms must occur at a specific phase of the menstrual cycle. The author discusses theories of ætiology, dismissing each popular hypothesis as lacking sufficient validation to account for every manifestation of the syndrome. He states that water retention is a clear-cut finding, but cannot be held responsible for the psychiatric symptoms. The association of low progesterone and high unantagonized œstrogen levels in the premenstrual phase has not been definitely proved. Abnormalities in vaginal smears, basal temperatures, the results of endometrial biopsy and urinary hormonal assays have not been correlated closely with the syndrome. Change in the blood glucose levels is often implicated, but the author comments that subclinical hypoglycæmia (which may occur in premenstrual tension), is a reflection of many different states and is not a cause of these states. Referring to the relationship of mental symptoms to premenstrual tension, he emphasizes that the emotional reactions to stress depend on the personality and not upon the stress. He considers it is reasonable to anticipate that any emotional symptoms, especially the "neurotic" variety, would be aggravated during this period. The brief literature on the relationship of premenstrual tension and mental disease is reviewed, and the author concludes that since the premenstrual tension syndrome is so common, and since psychosis in association with it is so rare, it can hardly be said that one causes the other. The legal aspects of this disease are complex, particularly as laboratory tests are of little value in diagnosis. The author briefly discusses the difficulties of substantiating a defence of "temporary insanity" in crime committed in the premenstrual week.

Differential Effects of New Phenothiazine Compounds.

F. A. FREYHAN (*Amer. J. Psychiat.*, January, 1959) discusses the therapeutic implications of the differential effects of new phenothiazine compounds. Patients were selected for neuroleptic treatment on the basis of specified psycho-pathological symptoms, and drug assessment was based on the kind and degree of modification of "target symptoms". Ten phenothiazine compounds were studied, and all were administered intramuscularly at least for the first week. The author notes the concurrence of greater potency with higher frequencies of extrapyramidal reactions. Chlorpromazine showed the highest incidence of dizziness and drowsiness in the initial stages of treatment. Promazine proved the least effective drug in this series, and its tendency to produce convulsions was confirmed. Trifluoperazine possessed the highest potency of all the phenothiazine derivatives investigated, and caused extrapyramidal symptoms with great frequency. Mepazine produced very poor clinical results, and convulsions occurred with this drug. The author mentions akathisia, or *impatience musculaire*, among the extrapyramidal signs of these drugs, the patients complaining of an irresistible urge to keep moving; this symptom disappears with anti-Parkinsonian drugs. He did not find that the occurrence of Parkinsonism contributed to favourable therapeutic results, or that this symptom occurred in all patients if given a sufficient dose. When it occurred, Parkinsonism appeared early in treatment. Assessing the therapeutic effects, the author notes that reserpine has a higher rate of failures in this series than all the phenothiazine derivatives except promazine.

Effect of Hypnosis on Intraocular Pressure.

A. S. BERGER AND P. J. SIMEL (*Psychosom. Med.*, July-August, 1959) have attempted to study the effect of hypnosis on intraocular tension with seven glaucomatous patients and four young healthy subjects. Four of the glaucomatous patients and all the healthy subjects could be hypnotized to the point of amnesia in one session. The most impressive finding was that on direct waking suggestion of symptom relief, all glaucomatous patients showed a drop in the pressure of one or both eyes to a level as low as (in one case) or lower than (in six cases) the lowest tension recorded during the previous year, during which they had been followed in the eye clinic. With post-hypnotic suggestion of symptom relief, several patients volunteered that they had fewer headaches and less tearing, felt generally more relaxed and slept better.

Electrocardiographic Alterations Observed during Percutaneous Cerebral Angiography.

J. A. EPSTEIN, I. HOFFMAN AND B. S. EPSTEIN (*A.M.A. Arch. Neurol. Psychiat.*, February, 1959) took serial electrocardiograms while cerebral arteriography was being performed, and the tracings showed slowing of sinus rate and sinus pauses, followed by nodal escape and the development of nodal rhythm for as long as 30 seconds. This suggested that the basis was vagal stimulation. Local infiltration of the area of the carotid sinus with procaine effectively eliminated the vagal responses previously obtained, suggesting that the afferent impulses responsible for stimulation of vagal centres in the medulla had their origin in the chemically sensitive carotid body. These results indicate that the morbidity associated with angiography may be related to the cardio-vascular effects of the dye. The authors conclude that the area of the carotid bifurcation should be anæsthetized before the performance of angiography in those patients in whom a fall in blood pressure or a change in cardiac rate or rhythm would be dangerous.

Acetylcholine and Migraine.

E. C. KUNKLE (*A.M.A. Arch. Neurol. Psychiat.*, February, 1959) has investigated cases of migraine by performing a lumbar puncture on the patients at the time of their attack, and by the use of a sensitive and specific bioassay method, an acetylcholine-like substance was detected in the fluid of five out of nine patients. No such substance was found in specimens from a group of 28 patients with extracranial vascular headache or headache due to muscle contraction, nor was the substance demonstrated in the cerebro-spinal fluid from patients with other neurological disorders. The author states that these results are consistent with, but do not prove, the hypothesis that acetylcholine or a closely allied substance is an active participant in the mechanism of migraine headaches, and that this suggests the use of atropine to modify the headache or prevent it.

Central Pontine Myelinolysis.

R. D. ADAMS, M. VICTOR AND E. L. MANCALL (*A.M.A. Arch. Neurol. Psychiat.*, February, 1959) describe central pontine myelinolysis as a new disease, diagnosed in four cases, in which the myelin sheaths of all the nerve fibres in the central part of the basis pontis had been destroyed in a single, large, symmetrical focus. The nerve cells and axis cylinders were spared for the most part, and the blood vessels were patent and unaffected. There were no signs of inflammation in or near the lesion. The disease had occurred on a background of alcoholism and malnutrition. The lesion is unique both in distribution and in the apparent character of the pathological process. It does not resemble any of the known varieties of demyelination and is differentiated from vascular diseases of the brain stem, Wernicke's disease, Marchiafava-Bignami's disease and from multiple sclerosis and related disorders. Two of the patients presented a dramatic clinical picture consisting of a rapidly evolving flaccid quadriplegia with weakness of the face and tongue and an inability to speak and swallow. In two other patients the lesion was apparently too small to produce clinical signs.

Congenital Lumbar Extradural Cysts.

G. W. SMITH AND M. CHAVEZ (*A.M.A. Arch. Neurol. Psychiat.*, October, 1958) discuss congenital lumbar extradural cysts. They are exceedingly rare and represent the least common cause of spinal cord pressure. In cases of spinal extradural cysts in the thoracic region the patient is usually an adolescent, with a history and symptoms of progressive spastic paraplegia. Pain is absent or is not a prominent symptom. Manometric tests demonstrate subarachnoid block with the characteristic spinal fluid changes of cord compression. Measurements on antero-posterior X-ray films show that the interpedicular spaces of the vertebræ

are enlarged. In cases of cysts of the lumbar region, the patient is more often an adult in his third or fourth decade, and the duration of symptoms is more protracted, being measured in years, whereas the average duration of the symptoms of thoracic cyst is measured in months. The long duration of symptoms is explained by the cyst's being located in and compressing predominantly the cauda equina rather than the cord and tracts. The patient complains of back pain, and in the X-ray picture there is a widening of the interpedicular space, with thinning of the pedicle and lamina opposite the cyst.

Chemopallidectomy and Chemothalamectomy for Dystonia Musculorum Deformans.

I. S. COOPER (*A.M.A. Arch. Neurol. Psychiat.*, January, 1959) discusses the alleviation of dystonia musculorum deformans by chemopallidectomy and chemothalamectomy. This disease is a progressive, incapacitating disease of childhood, characterized by involuntary movements and deformities of the entire body, and may appear first at about the age of seven or eight years, with hyperkinetic manifestations, torsion movements of one of the extremities, such as in-turning of one foot while walking or writer's cramp, or twisting of the hand and the wrist, or a dystonic torsion of the neck and shoulder. In 16 advanced cases in which the patients were subjected to chemopallidectomy and/or chemothalamectomy, 13 showed marked alleviation and reversal of the involuntary movements and deformities of the disease, the relief persisting for more than two years in several cases.

Gamma Encephalography.

T. PLANIOL (*Presse méd.*, January 17, 1959) has given the name gamma encephalography to the pre-operative exploration of the brain by radio-isotopes. This consists of the determination of the radiation discharged by the brain after injection of serum albumin marked with I^{131}, and the method has been found completely harmless and able to be used on ambulatory patients. After four years of experience with the method, the author is able to set out the indications for the use of the technique. He states that when the presence of an intracranial lesion, particularly a newgrowth, is suspected, but is not confirmed by other tests on the ambulatory patient, the result of this test, whether positive or negative, has a 90% probability. In the neurological and neurosurgical field, the findings in the gamma electroencephalogram are a good guide to the best technique to use to clarify the lesion which it suggests. If the patient's condition forbids the use of the procedure suggested, gamma electroencephalography may ultimately prove sufficient to confirm the provisional diagnosis. The method is also of value in following the progress of patients who have undergone operation for a cerebral tumour; it may even reveal an early recurrence and indicate its exact location and extent. It is also an excellent means of estimating the results of X-ray or cobalt beam therapy of a cerebral neoplasm, or of the antibiotic treatment of a localized infectious lesion in the brain.

PHYSICAL MEDICINE AND REHABILITATION.

The Nylon Elastic Stocking.

E. C. KLEIN AND L. J. BRONSTEIN (*Arch. phys. Med.*, February, 1959) have investigated the efficacy of nylon non-rubberized elastic stockings in the treatment of patients requiring elastic support for the lower extremities. They state that many such patients will not wear elastic support unless it is esthetically suitable and economically reasonable. Patients in this group found that the nylon stockings fulfilled their requirements. The stockings provide adequate support in mildly to moderately abnormal venous states of the lower extremities causing œdema. Symptoms associated with varicose veins, the post-phlebitic phenomenon and chronic venous insufficiency are relieved to a significant extent by the wearing of nylon elastic stockings. A large number of patients with "tired feet", but no demonstrable abnormality, obtained symptomatic relief from the stockings. The elasticity and compressibility of the stockings were retained for at least two months. In all instances, patients who wore the stockings stated that their associates could not distinguish the stockings from those they usually wore. The stockings are not recommended in advanced venous disorders in which the venous pressure is high.

Ischæmic Ulcers.

M. KOSIAK (*Arch. phys. Med.*, February, 1959) has investigated the ætiology and pathology of ischæmic ulcers. Ulcers were produced in dogs by both high pressures applied for short periods and low pressures applied for long periods. The author states that the time-pressure relationship is inverse and follows a parabolic curve. Microscopic examination revealed pathological changes in tissues subjected to such small pressures as 60 mm. of mercury for one hour only. Skin and subcutaneous tissue normally exert a sling or suspension effect, with the result that only a fraction of the applied pressure is transmitted to the deep tissue. Because living tissue is so sensitive to the ischæmia produced by even very low external pressures, temporary relief of this pressure every few minutes is essential. The author relates these experimental results to clinical situations.

Poliomyelitis-Injured Muscle.

E. F. ADAMS AND G. C. KNOWLTON (*Arch. phys. Med.*, March, 1959) report the results of an electromyographic survey of 120 poliomyelitis-injured muscles, which were evenly distributed through the Lovett grades from zero to G or better. The incidence of fibrillation, voluntary polyphasic units, normal contour motor units and silence was tabulated on the basis of a 60-point examination of each muscle. The authors conclude that on the average there is satisfactory agreement between the electromyographic incidence of voluntary motor units, voluntary polyphasic potentials, fibrillation potentials and silent points, and the response to the manual muscle strength test of affected muscles. They suggest that an appraisal of the initial injury can be obtained from the sum of the incidence of electrically pathological points (voluntary polyphasic potentials, fibrillation and silence). They suggest also that the intensity of spontaneous recovery can be appraised from the ratio of polyphasic potentials to the total pathological points. This has been called the "polyphasic index", and is found to correlate well with the manual muscle grade. The authors remark in conclusion that considerable variation occurs in the incidence of the various electromyographic features studied in muscles of the same grade.

Therapeutic Wheel-Chair for Above-Knee Amputees.

S. G. FEUER (*Arch. phys. Med.*, March, 1959) describes a wheel-chair which can be adapted in selected cases for the prophylactic or therapeutic management of patients with above-knee amputation stumps, who are confined to bed or a wheel-chair. He stresses the importance of preventing flexion contractures and thus shortening the period of rehabilitation. He states that the chair may be used in the following conditions: (i) double above-knee amputations; (ii) post-operative complications; (iii) concurrent injury or disease in the other extremity; (iv) concurrent illness, such as heart disease or hypertension; (v) post-operative debility, usually associated with senility, nutritional disorders, anæmia, etc.; (vi) poor motivation (emotional instability, psychological trauma); (vii) any post-operative above-knee amputation, when it is desirable to get the patient out of bed as soon as possible (to prevent venous stasis in other limbs, pulmonary complications, psychological deterioration, etc.).

Moist Heat in the Treatment of Neck and Shoulder Pains.

Y. M. CORDRAY AND E. M. KRUSEN, JUNIOR (*Arch. phys. Med.*, March, 1959) have made a study of more than 100 patients with symptoms of pain in the neck and shoulder and headache, classified as "cervical syndrome", with the object of evaluating the use of "Hydrocollator" packs as opposed to short-wave diathermy in treatment. All the patients selected were treated with the moist heat packs as the sole source of heat; this was usually followed by deep sedative massage and an active neck exercise programme. Several of the patients were treated by "Hydrocollator" packs because short-wave diathermy was contraindicated. The series was divided into injury and non-injury groups, and further subdivided according to the ætiology. Therapeutic results for the injury group (48) were as follows: good improvement, 60%; moderate improvement, 29%; no improvement, 11%. In the non-injury group (45), the following results were recorded; good improvement, 42%; moderate improvement, 44%; no improvement, 14%. Of the group of seven patients with acute fibrositis, all but one showed good improvement. The authors state that the results obtained by treatment with "Hydrocollator" packs compared favourably with those obtained by short-wave diathermy. The major clinical advantage was the greater comfort of the patient.

British Medical Association.

TASMANIAN BRANCH: ANNUAL MEETING.

THE annual meeting of the Tasmanian Branch of the British Medical Association was held at the Royal Society's rooms, Hobart, on March 7, 1959, DR. A. MCL. MILLAR, the President, in the chair.

ANNUAL REPORT OF THE COUNCIL.

The annual report of the Council, having been circulated to members, was received and adopted on the motion of Dr. W. W. Wilson, seconded by Dr. M. W. Fletcher. The report is as follows.

The Council has pleasure in presenting the annual report for the year ending December 31, 1958.

Membership.

The membership consists of 260 members as against 259 members for 1957, a gain of one.

Obituary.

It is with regret that we record the death of the following member: Dr. Edward Brettingham-Moore.

Meetings.

The annual general meeting of the Branch was held on March 29, 1958, at the Royal Society rooms, at which there were 46 members present.

The following office bearers were elected:

President: Dr. A. McL. Millar.

Vice-President: Dr. R. A. Lewis.

Medical Secretary: Dr. K. Melville Kelly.

Honorary Treasurer: Dr. F. R. Fay.

President Elect: Dr. L. H. Wilson.

Committee members elected were: Dr. J. Correy, Dr. A. J. M. Dobson, Dr. H. M. Fisher, Dr. D. B. Nathan, Dr. W. W. Wilson, Dr. H. Gatenby.

No other meeting was held during the year, as all business was carried out at the monthly meetings of the Southern and Northern Subdivisions.

Branch Council.

Fourteen meetings of the Council were held, twelve in Hobart, and two in Launceston. Additional members of the Council were:

Federal Council Representatives: Dr. J. B. G. Muir and Dr. L. N. Gollan.

Secretary of Southern Subdivision: Dr. R. J. Hudson.

Secretary of Northern Subdivision: Dr. W. H. Hill.

Immediate Past President: Dr. M. W. Fletcher.

The following attendances were recorded:

Dr. M. W. Fletcher	12
Dr. K. M. Kelly	13
Dr. R. A. Lewis	12
Dr. A. J. M. Dobson	11
Dr. J. B. G. Muir	4
Dr. A. O. Green	1
Dr. K. J. Friend	3
Dr. L. N. Gollan	12
Dr. R. J. Hudson	11
Dr. H. M. Fisher	11
Dr. P. L. Dorney	3
Dr. L. Wilson	9
Dr. F. R. Fay	13
Dr. W. H. Hill	9
Dr. A. McL. Millar	12
Dr. R. Wall	1
Dr. W. W. Wilson	9
Dr. H. B. Gatenby	6
Dr. J. Correy	11
Dr. D. B. Nathan	9

Representation and Subcommittees.

Federal Council.

The Branch was represented on the Federal Council by Dr. J. B. G. Muir and Dr. L. N. Gollan, both of whom attended interstate meetings on behalf of the branch. Dr. Muir unfortunately found it necessary to resign during the year due to ill health, and Dr. F. R. Fay was appointed in his place. Dr. M. W. Fletcher also attended one Federal Council meeting as proxy for Dr. Muir.

We wish to record the thanks of the Branch to Dr. J. B. G. Muir, who has retired after some years of service as our representative. He has ably carried out the not inconsiderable duties of the office.

Matters Dealt With by the Council.

Matters of importance dealt with by the Branch Council include:

Annual Representative Meeting.

Dr. K. J. Friend and Dr. P. Pitney were appointed to represent the Branch at the Annual Representative Meeting in Britain in July, 1958.

National Health Service.

Representations were made regarding anomalies of the medical benefits schedules, rates of payment for pensioners, higher hospital tables, benefits for patients with chronic disease, and many other minor items. Most of these items were discussed especially at Federal Council level, and this Branch has been active in support. Members will already have noted the improvement in conditions during the latter part of the year.

Australasian Medical Congress (Tenth Session).

The Branch has been congratulated on the success of the Congress, and there would seem to be no doubt that it was well up to the standard expected. Our thanks are due to the many members of the Congress Committee presided over by Dr. J. B. G. Muir, and to the Congress Secretary, Dr. Franklin R. Fay, for his excellent organization and untiring devotion to his mammoth task.

Conferences with the Minister for Health.

Representatives met the Minister on several occasions, and subjects discussed included views on health services, St. Helen's private hospital, cancer campaign, and home confinements. Other subjects are still under discussion, and an understanding of mutual problems has resulted.

Practice in New Housing Areas.

Talks were held with the Director of Housing with reference to difficulties experienced by some doctors in finding suitable surgery accommodation in new areas. As a result it is expected that these difficulties will be avoided in future.

Repatriation Department.

The Senior Medical Officer of the Department was interviewed on several occasions with reference to complaints of members concerning aspects of administration, and the position clarified after an exchange of views.

Building Subcommittee.

This year the building subcommittee has been very active, and several properties were inspected. It is expected that a building suitable for eventual use as a Branch headquarters will be obtained in the near future.

Notification of Births.

The Council took an active part in formulating the new form of notification of births, and it is hoped that the information made available will result in some improvement in the perinatal mortality rate.

General.

Many minor matters included advice to members on many subjects, information supplied to various inquiries by lay persons, and initiation of public health improvements.

Congratulations.

All members of the Branch were pleased to note that a Knighthood has been conferred on Dr. Ralph Whishaw, in recognition of his services to medicine in Tasmania.

Representatives.

The Branch was represented on outside bodies as follows:

Australasian Medical Publishing Company Limited: Dr. W. E. L. H. Crowther.

Road Safety Council of Tasmania: Dr. F. Phillips.

BRITISH MEDICAL ASSOCIATION (TASMANIAN BRANCH).

Income and Expenditure Account for the Year Ended December 31, 1958.

	£ s. d.		£ s. d.	£ s. d.
To Secretarial Fees	378 0 0	By Members' Subscriptions		2838 10 0
„ Printing and Stationery	44 6 9	*Less* Transferred Building Fund	418 0 0	
„ Postages	86 4 1	Post-Graduate Committee	104 10 0	
„ Code Address	3 3 0			522 10 0
„ Rental *re* Annual Meeting	2 0 0			2316 0 0
„ Travelling Expenses	262 10 0	„ Interest:		
„ Capitation Fees:		Commonwealth Bonds	49 1 6	
Southern Subdivision 111 15 0		Australasian Medical Publishing Company Limited	60 14 3	
Northern Subdivision 82 10 0		E.S. and A. Bank Limited	27 4 2	
Federal Council 311 5 0				136 19 11
Australasian Medical Publishing Company Limited 249 0 0		„ Car Badges		8 16 6
London: British Medical Association 426 3 9		„ Exchange		5 0
	1130 13 9			
„ Annual Audit	8 8 0			
„ Typing (Minutes, Newsletters, etc.)	189 10 4			
„ Freight	1 11 6			
„ Legal Expenses	28 17 0			
	2185 4 5			
To Surplus for year ending December 31, 1958	276 17 0			
	£2462 1 5			£2462 1 5

Balance Sheet.

	£ s. d.	£ s. d.		£ s. d.
Accumulated Fund Account:			Commonwealth Treasury Bonds	1327 5 0
Balance, December 1, 1957	2702 15 10		War Savings Certificates	133 0 0
Plus: Australasian Medical Publishing Company Limited	124 10 0		Australasian Medical Publishing Company Limited	
	2827 5 10		Debentures £1095 0 0	
Surplus for year	276 17 0		Sydney (Cash in Hand) 77 6 3	
	3104 2 10			1172 6 3
Less Transfer to Building Account	16 10 10		E.S. and A. Bank Ltd.	460 18 3
		3087 12 0	Furniture	30 0 0
War Relief Contribution		2 11 0	Gestetner	95 0 0
Sundry Creditors				
Building Fund 61 10 0				
Less Architect's Fees 21 0 0				
		40 10 0		
Post-Graduate Fund		13 10 0		
London: British Medical Association		26 15 6		
World Medical Association		39 3 0		
Adams and Bennetto		8 8 0		
		£3218 9 6		£3218 9 6

Building Fund as at December 31, 1958.

	£ s. d.		£ s. d.
To Mortgage Investment	500 0 0	By Balance Headquarters, January 1, 1958	248 4 4
Architect's Fees	21 0 0	„ Members' Subscriptions, 1957 and 1958	908 0 0
„ Balance:		„ Mortgage Interest	51 10 10
Hobart Savings Bank £656 3 4		„ Interest, Hobart Savings Bank	9 18 2
General Fund 40 10 0			
	696 13 4		
	£1217 13 4		£1217 13 4
		By Balance in hand	£696 13 4

Audited and found correct, subject to our letter of February 24, 1959.

ADAMS AND BENNETTO, Chartered Accountants (Aust.).

Federal War Relief Fund, 1914-1918: Dr. B. Hiller, Dr. R. Whishaw and Dr. F. W. Fay.

Tasmanian Physiotherapists' Registration Board: Dr. E. D. McIntyre.

Tasmanian Health Education Council: Dr. H. B. C. Houston.

Federal War Relief Fund, 1939-1945: Dr. R. A. Godfrey-Smith, Dr. T. Giblin and Dr. Franklin R. Fay.

Committees.

Ethics: Dr. R. A. Lewis, Dr. J. B. G. Muir, Dr. Franklin R. Fay, Dr. L. N. Gollan, Dr. M. W. Fletcher and Dr. W. W. Wilson.

Newsletter: Dr. K. M. Kelly, Dr. J. Correy, Dr. R. J. Hudson.

Publicity: Dr. W. H. Hill, Dr. D. B. Nathan, Dr. L. N. Gollan, Dr. J. B. G. Muir, Dr. Franklin R. Fay, Dr. A. McL. Millar.

Workers' Compensations: Dr. Franklin R. Fay, Dr. A. O. Green, Dr. A. McL. Millar.

Medical Fees: Dr. L. N. Gollan, Dr. W. H. Hill, Dr. R. J. Hudson, Dr. J. B. G. Muir, Dr. F. R. Fay, Dr. A. J. M. Dobson, Dr. L. H. Wilson.

Building: Dr. A. McL. Millar, Dr. F. R. Fay, Dr. K. M. Kelly, Dr. F. W. Fay, Dr. A. O. Green and Dr. D. J. Walters.

K. M. KELLY,
Honorary Medical Secretary.

Financial Statement.

The financial statement, having been circulated to members, was adopted on the motion of Dr. A. C. Corney, seconded by Dr. J. F. Correy.

The Treasurer, Dr. F. R. Fay, explained points of interest in the financial statement, and gave a résumé pertaining to the financing of the property in Davey Street for Branch headquarters.

Induction of President.

Dr. A. McL. Millar introduced the incoming President, Dr. L. H. Wilson, and vacated the chair in his favour. Dr. Wilson thanked the members for his election as President of the Branch, and moved a vote of thanks to the retiring President, Dr. A. McL. Millar, for his work as President of the Branch during the past year. This was carried with acclamation.

Election of Officers.

It was announced that the officers of the Branch for 1959 are as follows:

President: Dr. L. H. Wilson.

Vice-President: Dr. W. W. Wilson.

President-Elect: Dr. R. A. Lewis.

Treasurer: Dr. K. J. Friend.

Medical Secretary: Dr. K. Melville Kelly.

Councillors: Dr. A. C. D. Corney, Dr. R. A. Godfrey-Smith, Dr. N. B. Nathan, Dr. M. W. Fletcher, Dr. K. S. Millingen, Dr. A. J. M. Dobson.

Federal Representatives: Dr. L. N. Gollan, Dr. Franklin R. Fay.

Secretaries of Subdivisions: South, Dr. J. F. Correy; North, Dr. W. H. Hill.

Appointment of Auditors.

Messrs. Adams and Bennetto were reappointed auditors for 1959.

Amendments to Rules.

Dr. Franklin R. Fay moved the following motion, of which requisite notice had been given:

> That rule 13 (c) be amended so that the ordinary subscription shall be increased by two guineas (£2 2s.) *per annum* for members of the Southern Sub-Division and one guinea (£1 1s.) *per annum* for members of the Northern Sub-Division and this amendment to be in force for a period of ten years only.

This was seconded by Dr. A. McL. Millar.

After discussion of this motion, Dr. Fletcher expressed the view that there would be no dissension by members of the Northern Sub-Division to payment of £2 2s. and in support of this gave notice of motion that he would move that Northern members pay the same amount as those of the South. Dr. Fay suggested that this be done at a special meeting to be called for this purpose during the post-graduate week-end, to be held at Launceston in November. This was agreed to.

The President then put the motion submitted by Dr. F. R. Fay, which was carried.

Donations Towards Building Fund.

The following donations were received towards the Building Fund: Dr. L. H. Wilson, £26 5s.; Dr. H. M. Fisher, £26 5s.

Dr. L. N. Gollan advised the meeting that Northern Sub-Division had agreed to contribute £100 towards the Building Fund.

The Tasmanian Post-Graduate Committee in Medicine.

The report of the Tasmanian Post-Graduate Committee in Medicine was read and received. Dr. W. W. Wilson, secretary of the Committee, explained the difficulties that had been encountered with regard to obtaining lecturers because of lack of finance, but said that, as the Committee was now assured of a regular income, arrangements could be made to ensure that visiting lecturers were invited to Tasmania.

Retiring President's Address.

Dr. A. McL. Millar then delivered his retiring president's address (see page 689).

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

INFLUENZA: WHAT IS IT NOW?[1]

[From the *Australasian Medical Gazette*, September 15, 1894.]

ALMOST since the winter began this year we have been informed day after day of the occurrence of cases of *influenza*: many deaths have been attributed to its wonderful and mysterious influence, and now the country has taken up the cry as well. Every case of a catarrhal affection of the respiratory organs from the nose to the pleura, be it mild or severe, is set down to *influenza*. If there is whooping cough in a district, and a person therein dies of broncho-pneumonia, that is reported as a case of *influenza* and everyone is satisfied. We, however, are not at all satisfied upon this point, namely, as to whether epidemic influenza, in cases in which Letzerich found his *Micrococcus Influenza* (commonly called "*La Grippe*") which since 1885 has ravaged Australia, Asia, Europe, and America, is now or has been existent in the State of New South Wales this winter. If any of our now numerous bacteriologists can come forward and demonstrate the influenza bacillus, which has recently been discovered by Pfeiffer, Kitasato and Canon, in many cases diagnosed as influenza, but bearing only a remote resemblance to that very pronounced disease, as seen in various parts of the colony, we will certainly accept his dictum. The newly established Board of Health Laboratory should take this matter in hand, for, if the diseases we have seen all this winter and which we have diagnosed as ordinary nasal catarrhs, pharyngitis, laryngitis, bronchitis and broncho-pneumonia, are all simply *influenza*, then it is clear that isolation, disinfection, and a rational germicidal therapeusis should be practised. Let us ask ourselves, has this hitherto been done? To our certain knowledge it has not: these cases have been spoken of in an off-hand manner as "influenza", but have not been treated as such, but the ludicrous side of the picture appears when the patient gets well in spite of the treatment, so to speak. In other words, the ordinary routine treatment of acute affection of the respiratory organs is instituted in such cases, without regard to the influenza part of them, and they get well, whereas if the dread epidemic, "*La Grippe*", was really about, the treatment ought, *ceteris paribus*, to be chiefly acute influenzal, if we may be permitted to coin a term, and the ordinary methods of treatment, as practised in the old country from

[1] From the original in the Mitchell Library, Sydney.

time immemorial (for there such are the winter diseases of every-day occurrence), ought to be useless and ineffectual. We fancy it would be of great service to the profession in New South Wales to have this matter settled once and for all. The points most worthy of discussion would be these. Is influenza (*"La Grippe"* the epidemic) now existent in New South Wales? What does the profession understand by the term "Influenza"? If "Influenza" occurs every winter here must it not cease to be considered an epidemic: is it not, ipso facto, endemic?

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

ANNUAL SUBSCRIPTION COURSE.

Professor Ray Farquharson.

PROFESSOR RAY FARQUHARSON, Professor of Medicine, University of Toronto, Ontario, Canada, will visit Sydney from June 6 to 14 as the Category A Lecturer for 1959 of the Australian Post-Graduate Federation in Medicine. Professor Farquharson will give the following lectures in the annual subscription course: Monday, June 8, 8.15 p.m., Stawell Hall, 145 Macquarie Street: "The Value and Dangers of Steroid Therapy"; Tuesday, June 9, 11 a.m., Students' Lecture Theatre, St. Vincent's Hospital, Darlinghurst: "Disorders of the Thyroid Gland"; Wednesday, June 10, 2 p.m., Maitland Lecture Theatre, Sydney Hospital: "The Types of Hypertension that may be Relieved by Treatment of a Primary Lesion"; Thursday, June 11, 11 a.m., Students' Lecture Theatre, The Royal North Shore Hospital: "Medical Diseases of Bones"; Friday, June 12, 1.15 p.m., Scot Skirving Lecture Theatre, Royal Prince Alfred Hospital: "Disorders of the Pituitary Gland".

Dr. John J. Conley.

DR. JOHN J. CONLEY, Chief of the Head and Neck Department, Pack Medical Group, and Chief of the Head and Neck Service, St. Vincent's Hospital, New York, will visit Sydney from May 30 to June 12, 1959. The following programme of lectures, case demonstrations and operations has been so arranged that all cases for operation will, as far as possible, be demonstrated and discussed on the day before operations. Except where otherwise indicated, the programme has ben arranged at St. Vincent's Hospital. By arrangement with the New South Wales State Cancer Council, the lectures and case demonstrations will be free to all medical practitioners.

The key to the sphere of interest of various groups at the lectures and demonstrations is: 1, oncologists; 2, general surgeons; 3, otolaryngologists; 4, plastic surgeons; 5, dermatologists; 6, dentists; 7, radiologists.

The academic and operative programme is as follows:

Monday, June 1: 2 p.m., Students' Lecture Theatre, lecture and case demonstrations: "Carcinoma of the Tongue and Floor of the Mouth—Reconstruction" (1, 2, 3, 4, 7); 8.15 p.m., lecture, Stawell Hall, 145 Macquarie Street: "Signiiicance of the Lump in the Neck" (1, 2, 3, 7).

Tuesday, June 2: 8 a.m., operating theatre, operations: "Carcinoma of Floor of the Mouth—Reconstruction".

Wednesday, June 3: 8 a.m., operating theatre, operations: "Carcinoma of the Tongue"; 2 p.m., Students' Lecture Theatre. lecture and case demonstrations, "Carcinoma of the Thyroid" (1, 2, 3, 7).

Thursday, June 4: 8 a.m., operating theatre, operations: "Carcinoma of the Thyroid"; 2 p.m., Students' Lecture Theatre, lecture and case demonstrations: "Carcinoma of the Antrum" (1, 2, 3, 4, 7).

Friday, June 5: 8 a.m., operating theatre, operations: "Carcinoma of the Antrum"; 2 p.m.: Students' Lecture Theatre, lecture and case demonstrations, "Malignant Tumours of the Ear and Temporal Bone" (1, 3, 7).

Monday, June 8: 2 p.m., Students' Lecture Theatre, lecture and case demonstrations, "Carcinoma of the Larynx and Pharynx with a New Operation for Vocal Rehabilitation" (1, 2, 3, 4, 7).

Tuesday, June 9: 8 a.m., operating theatre, operations: "Carcinoma of the Larynx and Pharynx with a New Operation for Vocal Rehabilitation"; 8.15 p.m., lecture, Stawell Hall, 145 Macquarie Street, "Treatment of Parotid Tumours and Facial Nerve Management" (1, 2, 3, 4).

Wednesday, June 10: 8 a.m., operating theatre, operations: "Parotid Tumours and Facial Nerve Management"; 2 p.m., Students' Lecture Theatre, lecture and case demonstrations, "Sarcomas in the Head and Neck" (1, 2, 3, 4, 7).

Thursday, June 11: 9 a.m., Students' Lecture Theatre, lecture and case demonstrations, "Mandibular and Maxillary Rehabilitation and the Use of Free Skin in Head and Neck Surgery" (2, 3, 4, 6); 2 p.m., Students' Lecture Theatre, lecture and case demonstrations, "Melanoma in the Head and Neck" (1, 2, 3, 5, 7).

Friday, June 12: 9 a.m., Students' Lecture Theatre, lecture and case demonstrations, "Treatment of Severe X-ray Dermatitis by Full Face Skin Replacement" (1, 2, 4, 5, 7).

COURSES FOR GENERAL PRACTITIONERS DURING JUNE, 1959.

The Post-Graduate Committee in Medicine in the University of Sydney announces that the following courses for general practitioners will be held during June, 1959.

Week-end Course in Rheumatic Diseases.

A week-end course in rheumatic diseases, under the supervision of Dr. Selwyn Nelson, will be held in the Scot Skirving Lecture Theatre, Royal Prince Alfred Hospital, Camperdown, between 10.15 a.m. and 5 p.m. on Saturday, June 6, and between 9 a.m. and 1 p.m. on Sunday, June 7, 1959. The course will take the form of case demonstrations, panel discussions and lectures on the following subjects: newer diagnostic methods; factors affecting prognosis in rheumatic diseases; pararheumatic diseases; mechanism and treatment of osteoporosis; the evaluation of newer steroids; special methods of treatment. The panel list will include the following: Dr. Naomi Wing, Dr. P. J. Benjamin, Dr. F. Harding Burns, Dr. Peter Harvey, Dr. Brian Haynes, Dr. A. Kitchener Smith, Dr. Ralph Reader, Dr. R. Robinson and Dr. John Sands. The feel for attendance at this course is £3 3s.

Week-end Course in Renal Diseases.

A week-end course in renal diseases, under the supervision of Dr. Ralph Reader, will be held in the Scot Skirving Lecture Theatre, Royal Prince Alfred Hospital, Camperdown, on Saturday and Sunday, June 20 and 21, 1959. The programme is as follows:

Saturday, June 20, chairman, Dr. T. M. Greenaway: 10.15 a.m., "Some Practical Aspects of Renal Physiology", Professor C. R. B. Blackburn; 11 a.m., "Acute Anuria", Dr. H. M. Whyte; 11.45 a.m., "Fluid and Electrolyte Therapy", Dr. L. W. Davies; 1.30 p.m., "Renal Calculus", Dr. H. H. Pearson; 2.15 p.m., "Obstruction of the Bladder Neck", Dr. M. S. S. Earlam; 3.30 p.m., "Renal Hypertension", Dr. R. B. Blacket; 4.15 p.m., "Renal Tract Infections", Dr. J. McDonald.

Sunday, June 21, chairman, Dr. T. M. Greenaway: 9.30 a.m., "Acute Nephritis", Dr. T. M. Greenaway; 10.15 a.m., "The Nephrotic Syndrome", Dr. R. H. Vines; 11.30 a.m., "Management of Chronic Renal Failure", Dr. Ralph Reader. The fee for attendance at this course is £3 3s.

Week-end Course in Medicine at the Rachel Forster Hospital.

The following week-end course in medicine will be held at the Rachel Forster Hospital for Women and Children, Redfern, and will be open to all members of the medical profession:

Saturday, June 27, Dr. Willa Nelson in the chair: 9 to 10.30 a.m., "Pain in the Arm": (i) "Orthopædic and Neurological Aspects", Dr. Claire Weekes and Dr. Nanette de Coek, (ii) "Cardiovascular Significance", Dr. Jean Palmer, (iii) "Radiological Aspects", Dr. Jean Edwards; 11 a.m. to 12 noon, "Management of Renal Failure", Dr. Reginald Walker; 12 noon to 12.30 p.m., "Psychological Aspects of Enuresis", Dr. Marjorie Gilchrist; 1.45 to 2.30 p.m., "Common Symptoms of Unsuspected Endocrinal Origin", Dr. Helen Taylor; 2.30 to 3.15 p.m., "Practical Points in the Treatment of Peptic Ulcer": (i) medical, Dr. Willa Nelson. (ii) radiological, Dr. Mary J. Cronin; 3.30 to 5 p.m., "Skin Manifestations of Systemic Disease", (i) Dr. F. Goldschlag, (ii) Dr. Helen Taylor.

Sunday, June 28, Dr. Willa Nelson in the chair: 9.30 to 10.45 a.m., "The Differentiation of Episodes of Acute Myocardial Ischæmia", Dr. Jean Palmer; 11 a.m to 12.30 p.m., "Symposium on Treatment", (i) "Hypotensives", Dr. Willa Nelson, (ii) "Diuretics", Dr. Nanette de Coek, (iii) "Steroids", Dr. Claire Weekes.

The minimum number of candidates is eight, and the closing date for applications is June 12. The fee for

attendance at this course is £3 3s., and limited accommodation is available for women graduates for Friday and Saturday nights at £2 2s.

DIPLOMA COURSES, JUNE-JULY, 1959.

A course in advanced medicine, suitable for M.R.A.C.P. candidates, will begin on June 1 and continue for a period of eleven weeks. The fee for attendance is 30 guineas.

A course suitable for candidates proceeding to the Primary F.R.A.C.S. examination will begin on June 8, and continue for approximately ten weeks. This course has been organized and arranged by the New South Wales State Committee of the Royal Australasian College of Surgeons. The fee for attendance is 25 guineas.

On June 8, a course for the D.P.M. Part I will begin, and continue for a period of eight months. The fee for attendance is 50 guineas.

On July 6, a course for Part II of the D.A. will begin and continue for a period of three months. The fee for attendance is 40 guineas.

A course for D.C.P., Group I, will begin on July 6 and continue for a period of seven months. The fee for attendance is 35 guineas.

On July 20, a course for Part II of the D.P.M. will begin and continue for a period of seven months. The fee for attendance is 60 guineas.

A course for Part II of the D.D.R. will begin on July 20 and continue for a period of eight months. The fee for attendance is 50 guineas.

METHOD OF ENROLMENT.

Written application should be made to the Course Secretary, The Post-graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 4497-8. Telegraphic Address: "Postgrad Sydney".

THE MELBOURNE MEDICAL POST-GRADUATE COMMITTEE.

PROGRAMME FOR JUNE, 1959.

Visit of Professor Ray Farquharson.

Dr. R. F. Farquharson, M.B.E., D.Sc., F.R.C.P., F.A.C.P., Professor of Medicine in the University of Toronto, will visit Melbourne from June 21 to July 4. The following programme has been arranged:

Evening lectures, at 8.15 p.m. in the Medical Society Hall: Monday, June 22, "The Value and Dangers of Corticoid Therapy"; Thursday, June 25, "The Types of Hypertension that May be Relieved by Treatment of a Primary Lesion"; Monday, June 29, "Medical Diseases of Bone"; Thursday, July 2, "Emotional Manifestations in Patients with Structural Disease".

Visits to hospitals: Tuesday, June 23, Prince Henry's Hospital; Wednesday, June 24, Repatriation General Hospital, Heidelberg; Friday, June 26, St. Vincent's Hospital; Monday, June 29, Alfred Hospital; Tuesday, June 30, morning, Alfred Hospital, afternoon, Royal Melbourne Hospital; Friday, July 3, Royal Melbourne Hospital, with lunch-time lecture to resident medical officers on "Jaundice in the Adult".

The evening lectures form part of the Committee's Annual Subscription course. Non-subscribers may attend at 15s. per lecture.

Course in Medicine at St. Vincent's Hospital.

The honorary medical staff of St. Vincent's Hospital, Melbourne, will conduct a post-graduate course in medicine, suitable for candidates studying for the degree of M.D. and the diploma of M.R.A.C.P. This course will run for six weeks from June 1 to July 11. Sessions will be held from Mondays to Fridays at noon, 2.15 p.m. and 3.45 p.m., finishing at 5 p.m., and on Saturdays at 9.30 a.m. The course will consist of lectures, case presentations and ward rounds, with special demonstrations in radiology, neurology, electrocardiography, medical ophthalmology, pathology, dermatology, biochemistry, etc.

Enrolments, together with the fee of £31 10s. should be sent to the Melbourne Medical Post-graduate Committee, 394 Albert Street, East Melbourne.

Course in Pathology Suitable for F.R.A.C.S. Candidates.

A course in pathology, suitable for candidates for F.R.A.C.S. and F.F.A.R.A.C.S., will commence at the University of Melbourne on June 24, and continue on Mondays and Wednesdays from 1.30 till 2.30 p.m. (Anatomy classes will commence at 2.30 p.m. from that date.) The course will conclude on August 3.

Enrolments, with the fee of £7 7s. should be lodged with the Post-Graduate Committee two weeks before commencement, on the Committee's special form.

Course in Psychiatry.

Psychiatry courses will be continued during June.

The following courses will be conducted by the Australasian Association of Psychiatrists at 8 p.m. at the Royal Melbourne Hospital, on Thursdays: from May 28 to June 18, four sessions on "Child Psychiatry"—course controller, Dr. W. S. Rickards; from June 25 to July 16, four sessions on "The Problem of the Intellectual Handicap in Children and Adults"—course controller, Dr. J. F. Williams.

The Department of Mental Hygiene will conduct a course on general topics from 8 p.m. till 10 p.m. on Tuesdays, at the Royal Park Receiving House.

Country Courses.

Ballarat.—On June 25, at Craig's Hotel, Ballarat, at 8 p.m., Dr. Ian McDonald will discuss "Dysmenorrhœa and Hormones in Gynæcology".

Albury.—The Post-graduate Committee in Medicine in the University of Sydney, in consultation with the Melbourne Medical Post-graduate Committee, will conduct the following course at Albury on June 20 and 21: "Recent Advances in Gastro-Intestinal Disorders", Dr. W. E. King; "Failure to Thrive in Infancy", Dr. H. N. B. Wettenhall; "The Scope of Modern Cardiac Surgery", Mr. Kenneth Morris; "Management of Diseases of the Thyroid Gland", Dr. W. E. King; (*a*) "The Surgery of Arterial Disease", (*b*) "The Management of Chest Injuries", Mr. Kenneth Morris; "Some Aspects of Endocrine Disorders in Childhood", Dr. H. N. B. Wettenhall.

Dr. Ross Hayter, of the Border Medical Association, 399 North Street, Albury, is the local secretary. Details of times of sessions will be announced shortly. Those who have paid an annual subscription to the Melbourne Medical Post-Graduate Committee are invited to the above-mentioned country courses without charge.

Flinders Naval Depot.

At Flinders Naval Depot, on June 17, at 2.30 p.m., Mr. R. K. Newing will discuss "Facio-Maxillary Fractures". This lecture is to be given by arrangement with the Royal Australian Navy.

PRELIMINARY ANNOUNCEMENT.

Dr. Robert Kark, Professor of Medicine, University of Illinois, Chicago, will visit Melbourne from August 2 to 7, as the Pfizer Lecturer for 1959. He will give two evening lectures on August 3 and 6, on his work on the nephrotic syndrome and glomerular lesions in pregnancy.

Information.

The address of the Melbourne Medical Post-graduate Committee is 394 Albert Street, East Melbourne, C.2. Telephone: FB 2547.

Medical Societies.

THE ENDOCRINE SOCIETY OF AUSTRALIA.

IN June, 1958, the Endocrine Society of Australia was established at a meeting held at British Medical Association House, 135 Macquarie Street, Sydney. After the meeting had adopted a constitution, a scientific session was conducted.

The first annual meeting of the Society will be held in the Medical School of the University of Adelaide, Frome Road, Adelaide, on May 25 and 26, 1959. The scientific programme is as follows:

Monday, May 25: 2.30 p.m., report of Council proceedings; 3 p.m., Presidential Address: "Oral Hypoglycæmic Compounds", E. Downie; 3.45 p.m., "Experimental Diabetes in

Ruminants", I. Jarrett; 4.15 p.m., "Metabolically Active Polypeptides of the Human Pituitary", J. Bornstein and P. Davoren (read by P. Davoren).

Tuesday, May 26: 9.30 a.m., "Cushing's Syndrome and Thymic Tumours", P. Taft; 10 a.m., "Steroid Levels in Cushing's Syndrome and after Corticotrophin in late Pregnancy", A. W. Steinbeck; 10.30 a.m., "The Effect of Treatment on Acromegaly with Special Reference to Œstrogens", R. Melick; 11.15 a.m., "A Comparison of the Early Metabolic Effects of Triiodothyronine and Thyrotropic Hormone in Man", B. S. Hetzel (in collaboration with J. Scharnock and B. F. Good); 11.45 a.m., "Thyroid Hormone Binding Proteins", D. Winikoff; 2.15 p.m., "Clinical Studies on Œstrogen Excretion in Cancer", R. P. Jepson (by invitation); 2.45 p.m., "Anti-Œstrogens", C. W. Emmens, R. I. Cox and L. Martin; 3.30 p.m., "Hyperparathyroidism—A Review of 14 Cases", H. Breidahl; 4 p.m., "Citrate Metabolism in Hyperparathyroidism", L. Watson.

Notes and News.

Address at Sydney by Dr. R. Castañeda.

A distinguished microbiologist, Dr. Ruiz Castañeda of Mexico City, has agreed to give an address to the Society of Experimental Biology at the Red Cross Blood Transfusion Service, 1 York Street, Sydney, on Tuesday, May 26, at 7 p.m.

Dr. Castañeda was associated some years ago with the late Professor Hans Zinsser at the Harvard University Medical School in their classical work on typhus fever. The stain he developed for Rickettsia is still known as Castañeda's stain. For the last fifteen years Dr. Castañeda has directed the research at the General Hospital in Mexico City. His main interests have been brucellosis and typhus. He has not indicated the precise title of his address on May 26, but it will be concerned with the host-parasite relationship in typhus and brucellosis, with special emphasis on the diagnosis and treatment of brucellosis, a subject on which he is an authority.

Opening of Faulding's New Research and Bacteriological Laboratories.

On April 28, 1959, the Premier of South Australia, Sir Thomas Playford, officially opened the new Faulding Research Laboratories and the Bacteriological Division at Torrenside, South Australia. Recently extended and equipped at a cost of £150,000, the new laboratories, staffed mainly by Australian university graduates, provide modern facilities for investigating new therapeutic substances and improvement to production methods.

The House of Faulding will have made 114 years of progress on May 19, and the managing director, Mr. A. F. Scammell, said that the company's progress and high reputation were due in no small measure to the individual and loyal service of its staff.

The Premier said that the research work being done by the House of Faulding was contributing to a great extent to the health and welfare of the people of Australia.

The official party was escorted over the new laboratories by the works manager, Mr. K. D. Johnson, accompanied by Mr. A. F. Scammell and Mr. M. R. Lodge (director), who thanked the Premier on behalf of the Board of Directors.

Congresses.

FIRST INTERNATIONAL SYMPOSIUM ON ANTI-INFECTIOUS AND ANTIMITOTIC CHEMOTHERAPY.

THE first International Symposium on Anti-Infectious and Antimitotic Chemotherapy will be held in Geneva, Switzerland, on September 12 and 13, 1959. The Patrons will be Professor E. B. Chain (Italy), Professor D. Bovet (Italy) and Professor S. A. Waksman (U.S.A.). The President is Professor G. Bickel (Geneva), and the Vice-Presidents and committee members are distinguished authorities from a number of countries. There will be three sessions, as follows: (i) sulphonamides-retard; (ii) antibiotics; (iii) antimitotics. An interesting panel of speakers

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED APRIL 18, 1959.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	2(1)	1(1)	7(4)	..	1(1)	..	..	..	11
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	5	..	..	..	..	..	..	..	5
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	1	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	4(4)	11(10)	4(3)	..	..	..	..	1	20
Diphtheria	..	..	..	..	..	..	..	..	..
Dysentery (Bacillary)	..	1	3(3)	5(5)	..	..	..	..	9
Encephalitis	2(2)	1	..	..	..	..	..	..	3
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	1(1)	..	..	2
Hydatid	1	..	..	..	..	..	..	..	1
Infective Hepatitis	65(22)	20(14)	3	10(6)	3(3)	1	3	..	105
Lead Poisoning	..	..	1	..	..	..	..	..	1
Leprosy	..	..	1	..	4	..	..	..	5
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	..	..	..	..	..	..	..	..	..
Ophthalmia	..	..	..	..	1	..	..	..	1
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	1(1)	..	..	..	..	..	..	..	1
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	14(8)	1(1)	5(2)	2(1)	..	..	2	24
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	12(5)	24(17)	3	4(2)	3(2)	1(1)	6	..	53
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1	..	..	..	..	..	1
Trachoma	..	..	..	..	3(1)	..	350	..	353
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	16(15)	9(6)	17(9)	18(11)	5(4)	1	..	..	66
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	1	..	1(1)	..	..	..	2
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

has been arranged, and the papers and discussions will be published in a special number of *Antibiotica et Chemotherapia*. The official languages of the symposium will be English, German and French, and there will be a system of simultaneous translation. Applications to attend the symposium should be addressed to the Secretary, Dr. P. Rentchnick, Case Stand 471, Geneva.

Medical Appointments.

THE undermentioned appointments have been made at the Royal Adelaide Hospital, Adelaide:

Dr. J. P. Madden has been appointed Honorary Urological Surgeon (with status of Honorary Assistant Surgeon).

Dr. L. J. Rice has been appointed Medical Officer, Night Clinic (Female).

Dr. F. K. Weston has been appointed Assistant to the Director, Psychiatric Clinic.

Dr. D. A. Hicks has been appointed Honorary Assistant Physician in Charge, Diabetic Clinic.

Dr. S. C. Milazzo has been appointed Honorary Visiting Medical Officer, Northfield Wards (with status of Honorary Assistant Physician).

Dr. A. H. Finger has been appointed Honorary Clinical Assistant, Dermatological Department.

Dr. B. D. Gillam has been appointed Honorary Clinical Assistant, Dermatological Department.

Royal Australasian College of Surgeons.

MEETING OF THE NEW SOUTH WALES STATE COMMITTEE.

A MEETING of the New South Wales State Committee of the Royal Australasian College of Surgeons will be held on Wednesday, May 27, 1959, at 8 p.m., at 145 Macquarie Street, Sydney. The subject for discussion is "Carcinoma of the Antrum", and the speakers will be Mr. A. B. K. Watkins, Mr. N. C. Newton and Mr. L. Atkinson. All medical practitioners are invited to be present.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Lemech, Lubomyr, M.D., 1947 (Univ. Innsbruck) (registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act*, 1938-1958), 30 Heath Street, Wagga Wagga.

Vall, Leslie Joseph, M.D., 1932 (Univ. Budapest) (regional registration for the Coramba region under the provisions of Section 21A of the *Medical Practitioners Act*, 1938-1958), Gale Street, Coramba, New South Wales.

Compton, Ronald Murray, M.B., B.S., 1956 (Univ. Melbourne), 13 N.S. Trg. Bn., Ingleburn.

Szabo, Aladar, M.D., 1915 (Univ. Budapest) (registered in accordance with the provisions of Section 17 (2A) of the *Medical Practitioners Act*, 1938-1958), 102 Birriga Road, Bellevue Hill.

Kanarek, Maksymilian, M.D., 1951 (Univ. Cracor), regional registration for Urbenville region, Section 21A), Boomi Street, Urbenville, New South Wales.

The undermentioned has applied for election as a member of the Victorian Branch of the British Medical Association:

Gergely, Magda (née Feher), M.D., 1950 (Univ. Budapest), D.O. 1954 (Univ. Budapest) (temporarily registered in Victoria under Section 51 of the *Medical Act* of 1958), 58 West Toorak Road, South Yarra.

Large, Paul Garner, M.B., B.S., 1943 (Univ. London), M.S., 1952 (Univ. London), F.R.C.S., England, 1948, F.R.A.C.S., 1958, 386 Albert Street, East Melbourne.

Deaths.

THE following deaths have been announced:

MAZEL.—Dorothy Mazel, on May 3, 1959, at Wahroonga, N.S.W.

SCHALIT.—Moïse Aaron Schalit, on May 11, 1959, at Sydney.

Diary for the Month.

MAY 26.—New South Wales Branch, B.M.A.: Hospitals Committee.
MAY 27.—Victorian Branch, B.M.A.: Branch Council.
MAY 28.—New South Wales Branch, B.M.A.: Branch Meeting.
MAY 28.—South Australian Branch, B.M.A.: Scientific Meeting.
JUNE 2.—Western Australian Branch, B.M.A.: General Meeting.
JUNE 2.—New South Wales Branch, B.M.A.: Organization and Science Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales. Anti-Tuberculosis Association of New South Wales.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations, other than those normally used by the Journal, and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those of the list known as "World Medical Periodicals" (published by the World Medical Association). If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

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